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Dr. Bart Ostro, Supervisor, Air Toxicology and Epidemiology Section

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Dr. Henry Gong, Air Resources Board Member

Mr. Stan Hayes, Environ, API and WSPA

Mr. John Heuss, Air Improvement Research

Dr. Eliza Humphries, American Academy of Pediatrics and Health Networks for Clean Air

Dr. Allen S. Lefohn, API and WSPA

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Dr. Nathan Rabinovitch, Engine Manufacturers Association

Mr. Mike Roggee, California Manufacturers Tech Association

Mr. David Schonbrunn, Transportation Solution Defense and Education Fund

Ms. Debbie Shprentz, American Lung Association

Ms. Linda Weiner, American Lung Association

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PROCEEDINGS

CHAIRPERSON KLEINMAN: Well, good morning. Glad to see we still have some folks left. And we're going to begin with the public comments part of the presentations.

So I'm going to turn this over to Richard Bode to moderate that. And we'll get started immediately.

HEALTH AND EXPOSURE ASSESSMENT BRANCH CHIEF BODE: Thank you, Dr. Kleinman.

Just to let you know too that we changed the first agenda item at the request of several speakers who might have to leave early. So we're going to do our public -- the actual public comments -- oral comments first and follow that with staff responses to review of written comments and oral comments.

So first I'd like to have Stan Hayes.

MR. HAYES: Thank you very much. Could we see if we can -- so that I don't have to do butterfly stories here.

I am ready to go. Thank you.

Thank you all for letting me speak today. I appreciate this. Welcome to San Francisco, a beautiful sunny day, which is of course all we ever have here.

You have -- oops, we have no picture, which is not entirely a bad thing, I suppose.

MS. WYMAN: Just click on the right side of the

projector.

MR. HAYES: That should be the first. It's not on the screen.

Well, let me just vamp a little bit here. I'll wing it for a while here.

My name is Stan Hayes and I'm a principal with Environ International Corporation. Environ, as you may know, is a consulting firm specializing in human health and ecological risk assessment. My offices are in Emeryville, just a little bit down the road here today.

My interest in ozone goes back 15 years or more to when I directed an ozone health risk assessment nationally for the U.S. Environmental Protection Agency. And I did this in the context of the staff paper at the time.

(Thereupon an overhead presentation was Presented as follows.)

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MR. HAYES: A couple of a months ago some of my -- some of the folks that I know in the Western States Petroleum Association -- could we -- I guess it's not quite level. Sue, it's not quite on the screen. Some of the headings are important.

No, it's the top of the slide. The computer is cutting it off.

All right. Let me call your attention to the handouts this morning. Some of these will have somewhat more than the usual -- I think you need to...

All right. Let me sort of -- this at least will show you the main part of the presentation. And then we can talk about the headings, which are really just headlines.

Today I understand that the -- well, let me finish. A couple of months ago I was asked to take a look at the staff report prepared by folks here on behalf of the Western States Petroleum Association and the American Petroleum Institute. And I've done that and filed written comments. Today I'd like to elaborate a little bit on some additional things that I think are important, that I think are somewhat new.

You know, there's this joke about --

(Laughter.)

ADVISORY COMMITTEE MEMBER DELFINO: View slide show.

MR. HAYES: Try "view slide show"?

ADVISORY COMMITTEE MEMBER DELFINO: "View slide show".

MR. HAYES: I don't think that will do the trick.

Let's just go back to the slide show and we'll wing it a little bit. It's the screen settings that has

to be changed. That's all right.

HEALTH AND EXPOSURE ASSESSMENT BRANCH CHIEF BODE: Sue, where's your other projector?

MS. WYMAN: In the car.

MR. HAYES: All right. That's all right.

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MR. HAYES: Ah, nice. All right. Thank you very much.

Well, I know that staff has indicated that the basis for the proposal is primarily the chamber data. But there are a number of places within the document in Chapter 8 and Chapter 12, and certainly in Chapter 10 of the benefits analysis, some fairly clear and direct statements about mortality -- ozone mortality. There are a couple of places where the document says that a 3 percent increase in ozone translates into a -- I'm sorry -- there's a 3 percent increase in mortality per 40 ppb increase in ozone. So that's something new with this particular staff paper and something that I think deserves some serious attention. So that's what I'm going to talk about here today.

I'd like to recommend to you today what I think are some important additional analyses, research work that I think will help to explore and expand our understanding of the ozone mortality issue. Specifically what I'd like

to do -- and we're cutting off a little bit of the presentation here. But I'd like to recommend a further evaluation to ensure that the results we're seeing in the epidemiological studies for ozone mortality are reasonable and consistent.

There's some further epidemiological analyses, whose nature I'll describe in a minute, I'd like to recommend to you.

And, finally, I think that there is additional effort that I would recommend to you to reconcile the epidemiological results to the human chamber and the animal toxicological data, perhaps involving new research.

And, finally, I've got a close that talks a little bit about how I think we might be able to move forward from this point.

Could we have the -- well, I guess I can do it here. Here we are.

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MR. HAYES: Let's talk about each of these things.

First off, I'd like to talk about my first recommended additional analysis. And, that is, a more detailed evaluation to ensure the reasonableness and consistency of ozone mortality results. Much of what we have known about ozone's effect comes from PM studies

where ozone has been included as a confounder -- particular confounder. But ozone and PM are really quite different. And there's no guarantee that there's a similar -- or certainly the same or even a similar shape to the concentration response function. The effects of confounding in temperature and weather and seasonality co-pollutants are different with ozone than they are for PM. The characterization of personal exposure is more challenging in many ways for ozone than PM, which is also not without its challenges.

So the question I think we need to be asking ourselves is whether or not we've done enough to evaluate the reasonableness and consistency of the ozone mortality results.

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MR. HAYES: I -- now, this is going to be a problem. Is there any way -- the right-most axis on this figure is the important -- well, I'll go with it.

There was published in November in the Journal of the American Medical Association article by Michelle Bell and colleagues that looked at an ozone mortality effect in 95 cities as analyzed under the NMMAPS program. One of the questions I had with respect to those results was whether or not, as I would expect, there was some relationship between the magnitude of the mortality effect

they found and the degree of ozone -- and the severity of the ozone air pollution in the cities that they looked at and would intuitively expect that to be true.

What I got on this figure, which I'll build for you here, is 95 cities in the study arrayed at the bottom. Although technically only half are printed because of problems with my computer. What this shows, each of these bars represents the attainment status of that area, that city with respect to the one-hour standard, ranging from attainment on the bottom all the way to extreme at the top.

You see two red bars. Ah, this is great. Actually you can't see two red bars. You can see Los Angeles is an extreme nonattainment area. And if you could just peak around the corner somewhere near where that light is, you would see that there is the City of Honolulu.

Somewhere out over here is Honolulu. Well, suffice it to say -- and I think maybe just to complete the presentation here -- what I've done is I plotted -- what I have plotted is the point estimate of the ozone mortality effect expressed as percent increase for 10 ppb of ozone increase. This is from the Bell paper.

And what I found, to my surprise, I guess, is that -- this being a zero axis -- that of the 95 cities,

there was only one or them, Orlando, at the low side, but that saw a negative effect for ozone, which obviously makes some intuitive sense. Every other city saw a positive or slightly above zero point estimate of the mortality effect.

What puzzled me is why it is so that the Los Angeles point estimate of mortality is only about a quarter of what you would see in Honolulu. Put it another way, Honolulu's got four times greater incremental effect on mortality from ozone than does Los Angeles. Those are cities that behaviorally would seem similar. I'm puzzled as to why that would be. I can't explain this. There are a couple possible explanations. One is that a ppb is more important in Honolulu than it is in Los Angeles. That seems implausible because, although you can't see it, out over here the City of New York has an extreme -- I'm sorry -- a severe nonattainment area has the highest point estimate. But still the fact -- or it could be that there's something about the methodology, the way in which confounding factors are addressed that results in something that's not quite there yet.

CHAIRPERSON KLEINMAN: Excuse me, Stan.

MR. HAYES: Yeah.

CHAIRPERSON KLEINMAN: What isn't apparent on the slide that some of the people in the audience could see --

what's not apparent is that the right-hand axis, it's not total mortality that you're showing; it's the rate.

MR. HAYES: It's the rate.

CHAIRPERSON KLEINMAN: It's percent mortality per unit ozone.

MR. HAYES: Precisely, precisely.

CHAIRPERSON KLEINMAN: And so that is going to be a function of a lot of endemic factors in the various populations, right?

MR. HAYES: It is, indeed; it is, indeed. Although, as I say, for Los Angeles and Honolulu --

CHAIRPERSON KLEINMAN: Yeah, I just wanted to make sure that people understood that what you're showing is not total mortality, which my guess is might be proportional to the exposure.

MR. HAYES: Well, yeah. And I wish we could see the entire slide here. But, yeah, that's in fact the case. But still, what it raises in my mind is some questions about my own understanding of what's going on here. And that same understanding, or lack thereof, is shown by this -- is illustrated in this figure from the Gauderman paper in September, the New England Journal article on the children's health study.

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MR. HAYES: And while there may be many reasons

for this, I look at this and, perhaps along with the people who designed the children's health study, I too was surprised that ozone shows a flat curve. There is not an association that was found in the lung function deficit work done in the human -- in the children's health study that implicates ozone, although there are other reasons why it does, to be fair.

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MR. HAYES: So I suppose what that says to me is that more detailed evaluation I think is warranted here to better understand the seeming perplexities that I've referred to a moment ago.

I'd recommend to you, secondly, that further epidemiological analyses be conducted. And I think specifically -- I would suggest that those analyses allow for the possibility of more biologically complex ozone exposure response relationships, particularly at low concentrations.

I would suggest to you that there's a need for further analyses to look at additional more biologically complex metrics of exposure: Frequency of occurrence of high concentrations; multi-year high peaks; duration of respites in between episodes.

And, finally, I'd suggest that there's a need for further analysis to address more particularly

ozone-specific confounders or effect modifiers.

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MR. HAYES: Let me show you some data. This is from the work that I referred to earlier in which for the U.S. EPA we analyzed a lot of response data in an attempt to develop dose response -- or actually exposure response relationships that we used that were cited in the staff paper at that time.

First off let me caution you also. These are three Avol, Kulle, and McDonnell, who looked at healthy adults exercising heavily in chambers for short periods of time. So whether or not this bears any probative relationship to mortality is highly arguable. I'm not suggesting it does. I am suggesting that there's some biological complexity to these responses.

If you look -- and what we're plotting here is the fraction of the population that experienced in this case an FEV1 decrement of 10 percent. We see that with the Avol and McDonnell work that there is a distinct S shape to the curve. We see with the Kulle paper -- the Kulle results that there is what appears to be a kind of hockey stick. This is FEV1 decrements greater than 10 percent.

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MR. HAYES: Here's respiratory symptoms, same

studies. And, again, while you see real complexity to this, you still see for two of the studies a kind of tailing off S shape to the curve. And with Kulle a power law that just continues with some bump here to rise.

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MR. HAYES: Well, without trying to argue the analogy between these other measures of acute response and acute mortality -- they're different people, different mechanisms maybe at play, all sorts of differences that render the two separate -- we can see here in this chart the graphical representation of what the staff paper is asserting about the exposure response relationship for the mortality effect of ozone. Staff paper says 3 percent increase in mortality per 40 ppb of ozone. It's a log linear relationship. This is the implied dose response -- or concentration response function.

It's pretty close to linear, which I've shown here for comparison, particularly down at the concentration ranges of interest -- of practical interest to us. So in effect we're making a linear assumption about the concentration response relationship.

If there's any chance that there is any kind of an S shape to that curve or perhaps a square concentration response -- and, Mike, I think you mentioned that yesterday -- then what you have -- and this is an early

portion at the lower concentration portions of the curve -- that the S-shaped undershoots the linear and log linear and overshoots it later on. Where this inflection point is, where this point is, as a sort of as asymptote, I can't say. I don't know. The S curve shown here is totally hypothetical.

But it seems to me that there's no reason why the Epi studies couldn' be redone to assume a different underlying dose response model or concentration response model. Why not use a three-parameter logistic, which is what's shown here, feed the data in as to see what the data say. Let the data do the talking. There either is an S shape or there isn't. I don't want to prejudge it. It just seems from the evidence I showed you earlier, however sketchy it is, that it seems like we ought not to reject this and perhaps a number of other alternative shapes to the underlying concentration response curve.

Why that's important is that down at the low end here again, where we spend all of our time under conditions of attainment, between 40 and 70 or 80 or whatever the standard is to be -- that's where people spend all their time. And whether or not the curve is down here or up near the linear or log linear one makes an enormous difference when we total up the amount of premature deaths.

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MR. HAYES: So final recommendation with respect to further analyses, I think there's's a need, and I would commend for your consideration, a need for additional research to better reconcile the epidemiological results with human chamber and tox data. I think that particularly at low concentrations additional research is really, really needed.

Secondly, I think that more realistic exposure protocols that better represent what's going on in the atmosphere are needed along with additional endpoints that might point is toward a better understanding of what the mechanism of mortality might be if there is one.

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MR. HAYES: And then, finally, and then I'm done -- the first sentence up there for the audience reads, "Because available epidemiological evidence on ozone mortality" I don't think is ready yet for use in establishing either the level of the standard or the margin of safety because I don't think it's ready yet for drawing final conclusions regarding causation, I would strongly recommend that however difficult it is to come up with the research money, with all of the competing interests for it that there are, this work needs to be done.

I would finally suggest that pending the results of that additional research, that either you defer statements about ozone mortality in the staff paper or you suitably qualify them.

Now, I think given the concern that I have about some of this stuff and the need for the additional research to try to resolve some of the questions I'm raising, that I would suggest dropping the quantitative mortality estimates. Or at a minimum, if you feel like you need to keep them in the staff report, that they be treated as a sensitivity analysis in the same way that EPA has treated this same issue in the past.

And I also finally understand that this may be the first time in your standard-setting efforts -- perhaps you did this with PM as well, but prior to that I don't think that a benefits analysis such as that in Chapter 10 was included in the staff report. So I would suggest that there's no reason to delay moving forward the staff report portion dealing with their summary of the literature awaiting this additional research on mortality.

So one possible mechanism for doing that might be, to moving it forward while continuing to leave the door open on the ozone mortality question, is to separate the two in the two different documents. That's exactly the same way that EPA currently does it. No reason --

that's not a reason for doing it here. But a suggestion nonetheless.

So with that, I will close. And if anybody has any questions, I'd be more than happy to try to answer them. If not, thank you very much.

HEALTH AND EXPOSURE ASSESSMENT BRANCH CHIEF BODE: You know what I think we're going to do is we're going to try and change projectors real quick and see if we've got one that works better.

CHAIRPERSON KLEINMAN: In that case, while we're doing that, did anybody on the Panel want to make a comment?

Ralph.

ADVISORY COMMITTEE MEMBER DELFINO: Yeah, I mean a couple of comments.

I think what you pointed out -- I think what you pointed out, Mike, is quite critical, in that looking across these various cities and plotting the percent change in mortality is probably not representative of the true exposure response relationship across the geographic regions. Because, you know, if you go from the East Coast, Midwest, West Coast, Hawaii, there are different competing factors in each city, both nonpollutant demographic factors and other pollutants -- differences in the pollutant mix.

And the other issue about mortality I think that's quite critical is that this is only one outcome and in fact it's -- I think everyone could agree, it's probably the least sensitive outcome. I don't know who made that comment yesterday, but -- and that other outcomes that are more frequent and have large health impacts include hospital admissions and emergency room visits and other morbidity effects. And just to look at mortality I think truly underestimates the impact of any particular pollutant.

And I think actually the document that reviewed the time series studies was quite clear that most of these time series studies, including NMMAPS, have underestimated the effects of ozone by looking across the entire year. I suspect this plot is from the full year data, not summertime ozone?

MR. HAYES: It is, yeah. This is from what was published in the -- article.

ADVISORY COMMITTEE MEMBER DELFINO: Yeah. So I suspect If you look at the seasonally appropriate adjusted risk ratios, you wouldn't see -- you would see something entirely different. I don't know what you'd see, but I think that would be probably more appropriate to look at it in that manner and probably look at it -- as they did in the NMMAPS report, to also look at regional estimates

as well. And in our case it would be the West Coast would probably be the best way to look at it, during the summertime, during the warm season.

The other thing that -- the other thing -- I don't know about this sinusoidal curve. I'm not quite sure -- this is on the graph where you have the three parameter logistic. The criticism is whether there are effects below a certain threshold. And I thought again the review was very clear in finding those studies that have tested for thresholds, including the panel studies. And I can think of some of my panel studies in fact that we've done that. And most people in the field recommend that sensitivity analysis be done by very a simple procedure; that is, you drop the data for days where concentrations are above different thresholds, like 80 ppb's. And in those cases they've pointed out in the report, very frequently you see -- still see associations.

MR. HAYES: I don't think my argument was that I know the answer. I can't tell you whether there is or is not that S-shaped curve there. I can't tell where the break point is. I can't tell you where it asymptotes if there is one.

I do think though that because we have reasons, unlike PM, reasons with the chamber data to see, albeit in different endpoints and different exposure regimens in

different people, we have a reason to think that there's some more biologically complex mechanism that it's at least possible. And I think that when we do these Epi studies -- and I think it was true for PM, but I think it's more true even for ozone -- that when we pick off the shelf the statistical analysis packages that we use, embedded in them is an assumption about the concentration response function. And then I -- if you know nothing else, to pick it as was done with PM, I understand certainly why researchers would do that. But with ozone we know from the 20-plus years of human chamber data more about the response and the animal toxicological data as well.

And so I think there's every reason to believe that it's a complicated thing, that what's going on is biologically complex and we ought to do these analyses, if nothing else, just simply to satisfy ourselves that we're not oversimplifying a much more complicated problem.

ADVISORY COMMITTEE MEMBER DELFINO: But you put this in reference to the mortality studies. And I don't -- I really don't think the time series studies -- I mean it's certainly possible to try to come up with some kind of non-linear curve for those associations. But I think what you're suggesting is that the biological response might be sinusoidal or some other non-linear kind

of response. And that sort of analysis should come out of a clinical study.

The human chamber study or other experimental design may be a panel study with personal exposure estimates. Because you're really assuming a certain degree of precision and being able to come up with a correct biological model that you don't -- you just don't have in a time series data set.

MR. HAYES: One of the things that we also do with these Epi studies, and I certainly understand why, is we look at current air quality, because that's what we have. We look at historical air quality. But what we're talking about in the context of standard setting are concentrations that by definition are at or below the concentration level of the standard. That's the definition of attainment. And I think down in that low end of the concentration range, the .04's, the .05's, the .06's, .07's, I don't think there's a lot of information from the clinical data.

And I was very interested in what Dr. Adams mentioned yesterday about the work that -- and I don't know if it's published or not yet. But it was quite interesting. I really think that that sort of work that he's talking about over here -- or talked about yesterday is very, very informative and critical in our estimates of

risk, because that's where you, you know, you -- you spend -- people spend so many, many more hours at lower concentrations than they do at the peaks, by definition obviously. That it is what is going on there that really is the thing that matters. And whether or not the Epi studies are correct in looking at historical data and its association with some endpoint, the question isn't so much that as it is what will their response be under lower concentrations.

CHAIRPERSON KLEINMAN: Stan, I'm going to break off this discussion because we do have other folks?

MR. HAYES: One last -- don't leave -- I hope -- I hope you will not leave without providing your guidance to staff here on the kinds of additional research that you see that you think needs to be done. Doesn't have to be the stuff I recommended. But I think new work needs to be done and you're the guys to figure it out -- the people to figure it out.

CHAIRPERSON KLEINMAN: Thank you.

HEALTH AND EXPOSURE ASSESSMENT BRANCH CHIEF BODE: Allen Lefohn.

MR. LEFOHN: Good morning. My name is Allen Lefohn. I'm from Montana. It's going to be minus 35 degrees with wind chill tonight. Not here. In Montana.

(Laughter.)

(Thereupon an overhead presentation was Presented as follows.)

MR. LEFOHN: I was born and raised in L.A. I grew up in the fifties with the smog, at what, 450, 500 parts per billion, and remember days that I couldn't breathe very well playing baseball during the summer. I went to UCLA, undergraduate, and to Berkeley for Ph.D, and got my Ph.D with George Pimentel.

Next overhead please.

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MR. LEFOHN: Some of the scientific experiences I have had and are doing now: I'm lead author for the Air Quality chapter for the draft criteria document that will be coming out at the end of this month. I'm also a lead author for the Exposure and Dose Response Section and the Effects of Ozone on Vegetation for that document.

For the last 25, 30 years my focus of research has been on the effects of ozone and how to characterize hourly average concentrations in a way that are biologically meaningful for vegetation. In 1980 our work led us to publish many, many papers beginning in '80 on the importance of the peaks for vegetation and how to characterize it in the form of exposure metrics that focus on those peaks and the distributions.

In 1987, Dr. Milan Hazucha from UNC published a

paper. And EPA immediately called me up because the paper showed that again peaks were important but this time for human health, and a lot of the research I had done earlier on vegetation was relevant for the human health area.

I'm responsible for synthesizing the various ozone profiles for some of the clinical human health chamber studies that are in the staff report here, in particular working with Dr. Adams in his research and such at UC Davis.

I had published over 150 peer review papers and technical reports and was an executive editor of AE, Atmospheric Environment, from 1989 to 1999.

Next please.

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MR. LEFOHN: You have a very large amount of material that we supplied you, that Dr. Paul Switzer who I've worked with for the last of six years, from the Department of Statistics at Stanford University. And he specifically focused on the mortality and epidemiology, but I think a lot of that is relevant in terms of other biological endpoints.

And his conclusion was that the Epi evidence cannot be used to draw robust conclusions regarding the circumstances and magnitudes of ambient ozone mortality, in particular whether reported ozone effects are

causative. Without a clear understanding of the reasons for inconsistent effect estimates, one cannot rule out the possibility -- and this is very important -- that the ozone effect estimates are null artifacts.

Next please.

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MR. LEFOHN: And recently, as pointed out a few minutes ago, the Bell, et al., piece was published in JAMA. Those are -- that's the ordering of the 95 cities, with the red on the left being Hawaii. And the greatest road of risk per 10 ppb is at the bottom, and the least is at the top. And Los Angeles is very near the top. And to the right is the eight-hour average design values that I've determined for the 2001 to 2003 time period. And obviously some of the things that you've all been talking about other things such as associate demographic and other pollutants, et cetera, certainly could be impacting what's going on here.

But also notice that the confidence intervals are going through zero for many, many, many, many of those 95 cities. Now, at the very bottom there's a national average. So it is significant that simply because you're taking a lot of nonsignificant numbers and -- you have a lot and when you divide, you get significance. It doesn't mean that that is a meaningful number.

Next over here please.

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MR. LEFOHN: These are the numbers from Honolulu, Hawaii, for the eight-hour daily maximum concentration from 1991 to 2000. Please note they're very, very low. So the question here: Do we need to pay attention to the magnitude, because it was done for the entire time period.

Now, the interesting thing is, most -- we talk about season versus year. But in fact the way that EPA does report its data from the majority of cities across the United States, it is for the ozone season. In other words you're not going to find data, except for California and a few other states, for 12 months. And so you have seasonal data essentially for many of the places.

But this is a very low eight-hour daily maximum. It actually -- this city was the lowest of the 95 cities.

So there's certainly reasons one can say there are confounding influences, there are all kinds of other things. We still believe in the epidemiological results. It's just they're highly uncertain.

One other aspect which Professor Switzer has brought up is that you're looking at a modeling artifact.

Next please.

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MR. LEFOHN: Stratospheric ozone over Honolulu,

Hawaii, the elevation is three meters. On March 9th-10th 2004, NOAA flew over the area as part of research project. And I received the telephone call probably around that time asking if I had data for Honolulu, because they actually were tracking stratospheric intrusion and wondered how far it got to the -- if it got to the surface. It was seen at Monoloa over a 100 parts per billion. Certainly that is a trackable concentration at the high elevation Monoloa site. But down near the ground where you're not seeing over 100 ppb but in the 50's and 60's it's certainly a lot different.

Those are the two days which usually -- some people are tornado chasers. I'm a stratospheric ozone chaser. And so one of the things I look for when I look at surface are enhanced levels. But enhanced levels are not 100 parts per billion. They're 40 or 50 parts per billion, but constant, meaning you have two days', three days' worth without any break in the evening or the morning.

Next please.

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MR. LEFOHN: One of the things that one is using in the estimates for epidemiology is the center-city-type analysis, where you basically take all the monitors within a county and you average it, and you basically say, "This

is my number that I'm going to use for that county and I'm going to use that on a daily basis" in whether it's 8-hour, 1-hour daily max or 24 hour.

In order to make that work, with a linear model, you only need that the correlation coefficients are high. If you're dealing with a non-linear situation -- which Dr. Switzer argues that there is indications for non-linearity -- and in fact the JAMA paper with their results argues for it also -- is that the absolute concentration has to be small too among monitors. This analysis I did as part of the criteria document that -- we did it for 24 areas across the United States. And I have segregated the data for California. What it says is that the minimum correlation coefficient is fairly low for most of the areas that we're looking at. And low, while I know is a subjective thing, but the point is it's not .9, .8; it's .2, .3, .4, et cetera.

The max correlation of course is fairly high. But you have a range of correlations depending upon which days the data are among the pairs. In addition, on the right side, the last two columns, are the minimum P90's, which is the 90th percentiles of the differences of absolute concentrations. And all we're doing is arranging those in a percentile distribution and picking the 90th percentile. What it says is the minimum 90th percentile

runs fairly large actually. And the max is fairly large, 40, 50 parts per billion many times.

So what we're seeing here is that the correlation coefficients are not real high and the absolute concentration values are not real low in terms of the differences. In other words, some sites are going up and other sites are not necessarily moving in the same amount or same direction all the time. In addition, the absolute concentrations, they're not zero. So if you have a linear model, the correlation coefficient is not helping you achieve the assumptions that you've put in it. If you have a non-linear model the absolute concentrations become important and where the people are becomes important within the cities you're looking at.

Next please.

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MR. LEFOHN: A bottom-line concern about the use of the Epi data in the standard-setting process, at this time Epi results, my recommendation, should not be used to establish either the level or the margin of safety for the ozone standard. There's too much variability. As I've just showed you, some of the assumptions are just not being met, which may explain the heterogeneity among cities that we're seen for the 95 city study as well as we saw for PM.

The available epidemiologic evidence on ozone mortality cannot be used to draw robust conclusions regarding causation. And the ozone standard, my recommendation, should be based on results associated with human exposure chamber studies that apply realistic exposure patterns. And what I mean by realistic is the eight-hour square waves that were used at constant concentrations are rarely found in the United States, rarely.

Next please.

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MR. LEFOHN: Policy-relevant background. This is a direct quote right from the document itself. I want to differentiate between natural background and policy-relevant background. It's important. Within the range of concentrations due to such external or controllable sources those concentrations that may impact determinations of compliance with air quality standards or limit the potential for air quality improvements due to control programs have been defined in the document as policy-relevant background. This is not the same as natural background, as I just said.

Next please.

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MR. LEFOHN: There's a large variability among

global models on the attribution of the contribution of natural ozone to policy-relevant background. One global model that staff has focused on, which is the Fiore, et al., model, estimates that natural background ozone levels in four-hour afternoon average concentrations -- those are not hourly averages -- are in the 10 to 25 ppb range and never exceed 40 ppb. In other words, natural background will never exceed 40 ppb, never, never.

Next please.

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MR. LEFOHN: Staff states in its December 3rd, 2004, response to the comments: "Data on 19th century ozone concentrations measured in Europe and the U.S. (Bojkov, '86) show that spring peak ozone partial pressures were about essentially 30 to 50 parts per billion in the Midwestern U.S. and ranged from around 20 to 30 ppb in Europe."

Next please.

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MR. LEFOHN: I might mention that the Bojkov piece has been used over and over again to state that ozone was very, very low in the 19th century and was 19 parts per billion in Europe. Why 19 compared to the numbers that I just quoted? That was the annual average. Now we're dealing with daily max. But the daily maxes

that Bojkov used were from Linhof. And Linhof had 7-hour average values. They were daytime, 0700 to 1400, and nighttime, 2100 to 0700.

Next please.

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MR. LEFOHN: Therefore, if the 7-hour maximum average concentrations were in the 30 to 50 ppb range in the spring time, during pretty industrial times, the hourly average concentrations from 1871 to 1903 in Michigan had to be higher or equal to or greater, let's say, than 50 parts per billion.

Thus natural background, not policy level background, concentration levels appear to be higher than 50. The estimates for the range of policy-relevant backgrounds of course have to be greater than the background values estimated by the model sighted by staff. Once again, Fiore, et al., said background never exceeds -- never -- 40 parts per billion.

Next please.

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MR. LEFOHN: The removal of all anthropogenic emissions. One of my comments was: "In some of the modeling efforts to estimate natural background ozone concentrations within North America investigators removed all anthropogenic emissions of NOx, CO, and non-methane

hydrocarbons, including NOx emitted from aircraft and fertilizer, but not biomass burning."

ARB's response was: "ARB does not propose a projected all anthropogenic sources of ozone precursors in California could be eliminated." In other words, fertilizer adds to the amount of policy-relevant background.

Next please.

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MR. LEFOHN: The averaging time's important. And the point I was making is that in the document itself, the 15 to 35 parts per billion were probably long-term averages. And that if you're talking about the variability and the distribution and such, it's going to be higher than that. And staff's response is: "We agree that reading the long-term mean values presented in the staff paper as absolute maxima could be misleading." And it's going to be changed.

Next please.

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MR. LEFOHN: Bottom line. Because it appears the policy-relevant background levels are higher than 40 parts per billion assumed by staff, therefore the rollback estimates for the ozone concentrations are too optimistic and actually will occur slower than predicted. In the

model that Barry Larson and his staff have worked on, the higher the policy-relevant background, the slower the reduction will be in the mid-level concentrations.

The peaks will come down, but the rate of decline of the mid-levels are going to slow down in a drastic fashion, depending upon how high the policy-relevant background is above the assumed 40. A higher policy-relevant background will result in a greater slowing down, as I've said, in the mid-level. And, consequently, health benefits estimated by staff probably have been overestimated.

Next please.

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MR. LEFOHN: Human health effects. Experimental exposures of human volunteers to air pollutants under realistic varying exposure-controlled laboratory conditions have provided important information directly relevant to standard setting.

The important ramifications reported by Hazucha, et al., and Adams, et al., is that a non-linear dose response relationship is evident. This is similar to the research that I did 25 years ago on vegetation.

Next please.

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MR. LEFOHN: The higher hourly average

concentrations elicit a greater effect than the lower values in a non-linear manner. A major implication of a non-linear dose response relationship is at the same 8-hour average. It's the same 8-hour average, with different distributions of hourly average concentrations, will elicit a different adverse effect.

Now, I wrote a paper in 1993 that said you shouldn't use an 8-hour average because you're going to get inconsistent results. But prior to that I had written a paper that said if EPA continued to push for a 7-hour seasonal average for vegetation, the agency would in fact get inconsistent results with the same long-term mean giving different effects because the distributions are different.

Case in point, Colstrip, Montana, in the middle of nowhere in eastern Montana had a 7-hour seasonal of 43 parts per billion. Chicago, illinois, at the time -- or just outside Chicago in the county had 43 parts per billion. Same average, different distribution. Chicago had a lot more peaks.

Next please.

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MR. LEFOHN: Okay. For those that are interested in ozone from Asia, work that I'm doing now with NOAA, which is different than the aircraft stock, deals with

looking at sites from all over the world, remote sites, and asking the question: Are we getting increases in ozone in the southern hemisphere and the northern hemisphere? The Lawson Volcanic National Park is certainly not one of the pristine sites that I would normally pick for the study. However, because the focus of Jaffe, et al., and others have been on the Lawson Volcanic National Park, I carried that along in the study.

And the bottom line here is that you are seeing changes in the distributions. And these are showing the bins, the 10 ppb bins by month over the period 1988 to 2003. And the negative numbers are simply saying that you're losing low values because they're going up. In other words you have conservation of the concentrations. Now, some are going to go down from the top. And bins obviously at the bottom have to go up. You have a different process here of low end coming up, the bottom line being that it's not -- there were spring trends, but the spring trends are not in March. They're in April, May; and then summer, you get June and July.

The bottom line from what I'm seeing from the data is that, yes, indeed you are seeing trends during the period, which I agree with the authors. However, you have the Redding, California, and the, Anderson, California site that also are subject to very high levels of ozone

starting in April. And whereas the authors, Jaffe, et al., said that they're sure that Lawson has seen Asia because ozone does not form in high concentrations in the spring time, I think that's incorrect based on my experience of looking at your data.

Next.

Is that it? We're done.

Thank you.

HEALTH AND EXPOSURE ASSESSMENT BRANCH CHIEF BODE: John Heuss.

(Thereupon an overhead presentation was Presented as follows.)

MR. HEUSS: Thank you all. She's trying to get that set up.

I am John Heuss with Air Improvement Resource, a consultant to the Alliance of Automobile Manufacturers.

I'd like to thank you for the opportunity to provide these comments.

Pleased that our 16-page set of additional comments was distributed at the end of day yesterday. And I'm sure in the wonderful restaurants you all visited last night you had a chance to read it. Would certainly ask that you do read it and consider it before you come to closure on the document.

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MR. HEUSS: First subject I'd like to talk about is policy-relevant background. And it's not -- the title of the slide isn't showing, but I can go ahead. It's on the handout material.

We've been concerned for some time that ARB underestimated the background. In 1987 it was -- background was listed as 04 as a maximum. In 2000 when the SB 25 priorities were set, the same conclusion was made about background. And indeed in the review draft from last summer, claim is that background is basically a maximum of 04. But this is actually -- is now acknowledged a mean.

We submitted analyses and quite a few references in the year 2000 on this subject to ARB. In 2003, this material was resubmitted. We added another 17 references at that point because it was several year later. And of these 40 references, only 3 showed up in the draft. And they are those that relate to transport of Asian ozone. And we're concerned that this is kind of a selected view of the data to minimize the influence of background. And yet both the U.S. EPA and others have concluded that 40 parts per billion is now a useful kind of average background, but that peaks, extreme values of background, are considerably higher. And so we submitted quite a bit of information from these references, several lines of

evidence that peak background is actually at or near your processed 8-hour standard.

In the material that was distributed last night, we also have some information from a study by Winkler and Chock where they looked at national park information for both ozone, trace metals and other things to try to understand which sites are the cleanest in California. And I came up with Lassen, and Death Valley.

In the yearly peak 8-hour levels over the last decade, in both those sites, averages about 80 parts per billion. So both those sites would be substantially out of attainment of your state-proposed 8-hour standard.

Now, they looked at 5-day back trajectories for the highest ozone incidences at those sites. And in the material that we provided on page 4, there's a figure that shows the results from Lassen. And basically when they looked at these 5-day back trajectories, there were some piles on values use at the sites that were associated with trajectories that came over major urban areas. But there were also elevated ozone on days when trajectories show air coming from the northern Pacific without passing significance population centers.

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MR. HEUSS: We're pleased that the staff response to comments now acknowledges that 04 is an average

background. However, it doesn't address the issue of maximum background. And if you're setting an extreme value standard as you have for 1 hour and are proposing for 8 hour, you need to consider the extreme values of background.

Staff does acknowledge that there will be off-season ozone exceedances -- there will be exceedances out of ARB control. They need to put in the document more information about the maximum background.

Conclusion we draw is the proposed 8-hour standard is within the range of policy-relevant background.

There are a couple implications from this. First is when policy-relevant background is high, again not high all the time, but when it is leaves very little room for ozone -- for man's activities.

And, second, even if you were successfully able to control manmade ozone down to only a few parts per billion, there will still be exceedances of the 070 standard throughout California.

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MR. HEUSS: Second subject that I'd like to talk about is the need for an exposure/risk assessment. The exposure/exertion profiles in the clinical studies, they are within the range of human behavior, but they don't

mimic typical or average human behavior. And so to determine the risk you need to take a look at the probability of being outside, the probability of exercising heavily, the probability of doing all this at the time of high ozone and indeed at the place of high ozone. All these probabilities are involved.

In the U.S. EPA review in the late nineties they carried out an exposure/risk analysis using a probabilistic exposure model. They used concentration response functions from the clinical studies, as Stan Hayes showed you, and then they evaluated the risk from various alternative standards.

They also looked at Epi studies and used some of the associations there.

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MR. HEUSS: Both EPA and ARB, your reviews are directed at similar mandates. Both agencies have the same basic requirement to protect the public health with an adequate margin of safety. And both rely on the same basic set of data relating to ozone concentrations and activity levels where effects are documented.

So we urge ARB to carry out an exposure/risk analysis to evaluate both the proposal and alternative standards. We think this kind of risk analysis is an integral part of the scientific process of setting ambient

concentration standards at levels that are relevant to plausible exposures and still ensuring an adequate margin of safety.

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MR. HEUSS: Next I'd like to review the EPA decision on the 8-hour standard they set. EPA, the Clean Air Science Advisory Committee, which was similar to your committee except had maybe three times as many people on it so their might be, say, three epidemiologists and three clinicians, et cetera, they looked at the information from the exposure and risk assessment, they carefully considered at some length when the changes in the clinical studies should be considered adverse, and they looked at the epidemiological information.

The CASAC input administrator was that only one standard was needed, not two. And they preferred at 8 hour.

They also recommended allowing multiple exceedances for robust planning for the federal standards, attainment demonstrations have to be put together, and people have to figure out how many funds of the various precursors needed to be removed. And they thought based on experience, trying to do this with less robust targets, that it was difficult.

The administrator then considered a range of

possible standards: 07, 08, 09 ppm, and in each case with from one to five exceedances. She looked at the information, obviously the information provided by the public, the information provided by CASAC and others. She chose 08 ppm. She chose a more robust and stable target. Again, the three-year average are the fourth highest of those 8 hours.

The reasons given that 07 was not chosen were things like no one on CASAC recommended 07. The individual CASAC members were asked their opinions. A number of them suggested 08, a number 09, some said 08 or 09, some said it's a policy choice. But no one on CASAC recommended 07. And, indeed, the administrator pointed out that 07 was too close to background levels and would focus controls on non-anthropogenic sources.

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MR. HEUSS: I'd also like to talk a little bit about a third subject, the epidemiology.

One of the major findings to come out of the HER reanalysis of PM time series studies is that the Epi is more uncertain than previously thought. Both series have model selection issues, have now come out to be even stronger than previously thought. We're not talking about trying to understand air pollution health effects in a complex mixture where you have a wide variety of highly

correlated variables. It's a difficult problem.

Now, we're pleased to see that the staff acknowledges on page 48 of the response to comments that there is some non-zero probability the effects are not causal. And, indeed, we think that should be carried over as this information goes forward to the Board interpreting the Epi studies, that we're not really sure in every case whether there's causality or not. And again for various kinds of studies, various kinds of endpoints, the consistency and the strength of the data varies substantially. And I think much of that is already expressed in the document.

I would urge the AQAC to discuss this, particularly the limitations of using single pollutant models. I'm particularly happy that ARB has had a major effort in the Fresno asthmatic study to try to get into more detail about what actual exposures are occurring in terms of the various possible causal variables and trying to understand more in the analysis about what is causal and what isn't.

Also pleased that the staff acknowledges that it's possible that there be no benefits from the ozone reductions on page 12. However, staff goes on to say meta-analysis suggests that on average health benefits would occur.

So now we get to the issue of meta-analysis, which brings us back to the NMMAPS data. And we have three pages of discussion in our 16-page document and two figures that walk through the NMMAPS material. The three various NMMAPS ozone mortality analyses that would have been done including the latest by Bell, et al.

In the September comments we provided ARB, we included three plots of individual city NMMAPS results that came from the Johns Hopkins website. In each case at lag 0, lag 1, and lag 2 there was a very wide range of ozone associations in individual cities. And two of those plots are included as pages 15 and 16 of our material that was distributed last evening. And I'll show in a minute a little bit of that.

Again, in each case and individual days, there's a range from minus 3 percent to plus 3 percent change in mortality for a 10-part-per-billion increase in ozone. This is an implausibly wide range that includes both negative and positive results.

Also, both the original AM analysis by the NMMAPS group for ozone and the reanalysis that was provided in 2003 report negative associations overall in winter for ozone. The Bell, et al., paper doesn't have any winter-specific results.

And, finally, as shown in Figure 12-2 of the ARB

document, the overall association of ozone is not significant in multi-pollutant models. So we're concerned that it's, in our view, premature to interpret the NMMAPS associations as causal.

The other kinds of things that we point out in our information is that the strongest association is on day zero or the same day. Since ozone is suppressed overnight and in the morning in cities, peaks in the early afternoon to mid-afternoon, an association on the same day raises some questions about the temporality assumption where the ozone exposure should precede response.

Or on the other hand you can look at it as a strong same-day association implies a very, very direct, immediate effect of ozone on mortality. And, indeed, when we're looking at the kinds of concentrations and personal exposures that would be involved in the bell, et al., paper, there are only -- well, the average concentration for all the cities is 26 parts per billion. Now, this is as measured at the monitors.

But since the vast majority of people spend the vast majority of time indoors, their actual personal exposures, particularly those of the frail population where they would be at risk, either in their home or in a nursing home or a hospital, these kind of exposures are roughly half the exposures outdoors. And so we're talking

about the possibility of 10 or 15 parts per billion ozone causing immediate death to a portion of the population. And I find that implausible.

Other implausible findings from the Bell, et al., study is that the associations are highest in some of the cleanest cities, the associations are essentially the same for all causes of death -- all major causes of death, and they're essentially the same for all age groups. So for these kinds of reasons I think it's premature to interpret the NMMAPS associations as causal. Now, clearly in Chapter 10 you're probably going to be doing sensitivity analysis of various kinds. And I think the idea that effects may not be causal is one of the possible outcomes when we finally understand this. And I think that needs to be included.

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MR. HEUSS: I'd like to show a couple of the findings of NMMAPS. This is from the reanalysis in 2003. This is ozone mortality at lag zero. And you'll have to take a look. I don't think you can quite see what we're looking at here. But the percent change in mortality -- again this is for the combined results -- and at the left of the winter results would show a negative combined result. An overall combined result with the bell-shaped curve in the middle, which is slightly positive. And then

the summer results, which are more strongly positive.

Now, if you go back to the first NMMAPS study with the general additive model, ozone was the only one of the five pollutants that they looked at that in overall combined year-long data had no ozone association. That middle bell-shaped curve was essentially backed over the zero point. But there was also the negative association in winter and the positive in summer.

So the difference between the GAM modeling and the GLM modeling to shift the ozone association is slightly positive. As you remember, it also shifted the PM10 association down somewhat.

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MR. HEUSS: But if you look at the individual city results -- in this it doesn't show up extremely well -- but the figure is in the material that was provided last night, where we're plotting the individual city results, this is for lag zero, from the smallest association to the largest. And there are 80 cities in this plot. It's located on the screen here. But it is in page 15 of the material we provided yesterday. And it runs from minus 3 percent to about plus 3 percent. And, as others have mentioned, roughly about seven of these are statistically significant out of 80.

Now, there is consideration of the positive

associations as health effects. But there are also a large number of negative associations which would apply benefits from ozone exposure. And nobody believes that's real. But it kind of shows the overall extremely wide range of results in these Epi studies.

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MR. HEUSS: On the next plot we're looking at day two, which is more typical, where again there's a wide range of positive and negative associations. And it's implausible to me to this extremely wide range. Now, in the Bell, et al. paper they don't show you this wide range for the individual cities. They do a second stage Beijing analysis which essentially compresses everything to go from 0 to 1 percent. But the actual raw individual city data for the different individual lags in each case runs from minus 3 percent to about plus 3 percent for 10 ppb change in ozone. And I guess again that to me is an implausibly wide range for me to accept these associations at this point.

There clearly are so many associations in literature there probably are significant ozone health effects. But trying to understand the nature of these from the Epi studies is very difficult. They're inconsistent results for every endpoint. Some are somewhat more consistent than others. There are issues as

pointed out yesterday of thresholds. And clearly in the benefits analysis, you will be having some sensitivity analysis to get some sense of the range. I think in each case that the argument can be made that zero should be included as part of that range.

So we are at this point asking you to consider all this as you go forward.

Thank you very much.

HEALTH AND EXPOSURE ASSESSMENT BRANCH CHIEF BODE: Great.

Linda Weiner.

MS. WEINER: Good morning. Thank you for the opportunity to speak.

My name is Linda Weiner and I represent the American Lung Association of California. The American Lung Associations of the Bay Area and the Bay Area Clean Air Task Force. We're a coalition of over 20 groups in the Bay Area representing public health, environmental and transportation organizations including, among many others, the Union of Concerned Scientists, the Natural Resources Defense Council, the Sierra Club, and several local environmental justice groups, particularly Bay View/Hunters Point Community Advocates.

I'm here today to lend my voice in strong support of a new 8-hour average standard for ozone. As you heard

yesterday, a number of studies have shown that avarice health effects from ozone can occur in healthy young adults at the current level of .08 parts per million. We are therefore asking to lower the level of ppm to .07, the highest level that can be considered protective of public health including a margin of safety.

We're also asking that this standard not be exceeded, that rounding-up methods allowing concentrations to exceed the level of the standard as is done with federal ozone standards not be utilized. And, additionally, we ask that multiple exceedances of this standard should not be tolerated due to the public health risk evident at .08 ppm.

We also ask you to endorse the proposed more stringent -- as we ask you to endorse the proposed more stringent 8-hour standard, we also ask you to endorse retention of the 1-hour average standard of .09 ppm also not to be exceeded. Both standards are needed to provide protection against short-term peaks and longer term exposure that can contribute to respiratory irritation and lung function.

We also want to add an important point that is not indicated in the staff report. And, that is, that the proposed new standards are especially justified, given the somewhat conservative approach taken in determining

populations at risk. High ozone levels affect not just individuals who spend, quote, significant periods of time outdoors, but also affect people with asthma, seniors, children, and those people already suffering from bronchitis and emphysema; and equally important and additionally, low income communities and communities of color that are disproportionately located in areas with major sources of air pollution and toxic contaminants. So that the residents of these communities are at higher risk for lung disease from ozone exposure, also another important classification.

For example, in the Bay Area we have West Oakland and Bay View/Hunters Point that have very, very high levels of asthma. And as you may be aware, the prevalence, severity and mortality rates for asthma are much higher in these particular communities, particularly San Joaquin Valley where asthma prevalence rates are three times higher than the national average.

The research of the public health impact of unhealthy ozone levels is continually mounting. A number of recent studies indicate that children living at high ozone areas may be affected for life by pollution exposures, with a significant lag on lung function -- lung function growth as determined by the southern California children's health study, a very credible study.

Perhaps even more disturbing, a most recent study published in the Journal of American Medical Association published a landmark study linking exposure to ozone to a significant increase in premature death in cities across the country.

In closing, we would add that millions of Californians, literally millions, are at risk or suffering from impaired lung function, irritated respiratory symptoms, increased respiratory and cardiovascular hospitalizations, increased asthma attacks, and subsequent emergency room visits for asthma and increased school absences if current concentrations of ozone continue. Asthma is a leading cause in schools of chronic disease.

These serious health impacts not only result in shortened lives and worsened quality of life for children and adults, but also add up to substantial cost to individual and society for hospital visits, health care and medications to treat pollution-related illnesses. So there's also an economic issue involved.

For these compelling reasons we ask that the Committee adopt our recommendations to revise and strengthen the ozone standards to protect public health.

And I would add, in closing, that we have a letter that is in your packet that represents a number of organizations that feel strongly about this same issue.

And these partners include Environment California; Kirsch Foundation; obviously American Lung Association of California; Environmental Defense; National Parks conservation Association; Merced/Mariposa County Asthma Coalition; Fresno Metro Ministry; Medical Alliance for Healthy Air, Sierra Club California; and Community Medical Center. So with these strong partners and with the serious public health impact we ask that you respectfully consider our recommendations.

Thank you.

HEALTH AND EXPOSURE ASSESSMENT BRANCH CHIEF BODE: All right. Thank you.

You know, I'd suggest why don't we take about a five-minute break. We're going to try and fix that laptop so the slides will work.

And pass this on to you, Dr. Kleinman. Come back about five minutes?

CHAIRPERSON KLEINMAN: Five minutes.

(Thereupon a recess was taken.)

HEALTH AND EXPOSURE ASSESSMENT BRANCH CHIEF BODE: Okay. Our next speaker is Nathan Rabinovitch.

And I know most the speakers are doing this. Make sure you introduce yourself and who you represent too. That'd be great.

DR. RABINOVITCH: Good morning. My name is

Nathan Rabinovitch. And I'm a physician at National Jewish Medical and Research Center.

(Thereupon an overhead presentation was Presented as follows.)

DR. RABINOVITCH: I've been doing air pollution studies for the past five years, looking mostly at particulate, but also at personal exposures to particulate and ozone. And we were asked by the Engine Manufacturers Association to take a look at the staff recommendations. And we submitted a paper -- a 16-page paper to you guys. I hope you'll take a look at it. And I'll be happy to talk about that after this summary if you have any questions.

The California Air Resource Board proposed a revised 8-hour ambient air quality standard for ozone at 0.7 parts per million not to be exceeded. This staff proposal is based primarily on the results of several human exposure studies which have assessed symptoms and pulmonary function over multiple hours. Epidemiological studies reporting that low-level ozone is associated with increased morbidity and mortality in subsets of the population are cited as supporting evidence.

This paper reviews the primary studies that serve as a basis for the revised ozone standards and assesses their applicability and limitations. The review

identifies several significant limitations associated with using the chamber and epidemiology studies to support evidence-based decisions to alter current standards. Among those issues are the following:

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DR. RABINOVITCH: Chamber studies assess immediate changes in symptoms and pulmonary functions and compare these responses to free air exposure. These challenges are essentially unblinded because of the odor of ozone, and appear generally to utilize the highest of three PFT measurements without regard to ATS criteria for reproducibility. If so, these are essentially effort-dependent measurements which may not truly reflect any loss of lung function.

The transient changes in pulmonary function often would not meet the defined ATS criteria as adverse events, as they are not accompanied by symptomatology.

In general, both FEV1 and FVC decline concurrently without changes in the FEV1 over FVC ratio, unlike classical asthmatic or reactive airway responses where FEV1 decreases typically occur with little change in FVC. This implies a restrict process possibly related to poor inspiratory effort but not necessarily to changes in the airways themselves.

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DR. RABINOVITCH: This being the case, there is no clear link between these acute transient events and any chronic airway effects as there would be in, for example, asthma. Furthermore, because exposures are not titrated at lower doses, no threshold levels can be delineated from these studies.

The ozone exposure levels which are associated with these acute changes in chamber models are cited as relevant ambient concentrations likely to cause health effects in the population. Based on personal exposure studies it can be assumed that in general the ratio of personal ozone exposure to ambient concentration is considerably less than 1, due to significant indoor activity in the daytime. Subjects must exercise vigorously in the chamber model before any acute response is observed, even when exposures are considerably higher than present standards.

It is unclear whether this effective dose of ozone is reached by a significant proportion of the population who would have to exercise outside in the summer heat for four to five hours before this level of exposure would be achieved. In fact, children, who are more likely to play outside, appear to be relatively insensitive to acute ozone exposure. Nor is there any evidence that older subjects or those with chronic airway

diseases such as asthma are more susceptible to ozone in chamber studies.

Thus, there is a weak connection between the chamber model and health effects in the population because of the unusual nature of the acute response and the indirect link between chamber exposures and personal ozone exposures.

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DR. RABINOVITCH: Epidemiology studies results have been inconsistent especially in regard to vulnerable populations and the occurrence true adverse events as opposed to transient changes in pulmonary function at lower ozone concentrations.

Furthermore, there is no evidence that children, the elderly or individuals with chronic airway diseases are more susceptible to the effects of ozone in the chamber studies. In fact, chamber study results of children and the elderly show decreased susceptibility to ozone. Epidemiology studies examining health effects in children or asthmatics have been inconsistent, and the children's health study found little evidence that ambient ozone was associated with any progression in pulmonary function deficits in children.

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DR. RABINOVITCH: We acknowledge that ozone

exposure at certain levels potentially represents a threat to the health of all or to a subset within the general population. However, considering the absence of definitive data demonstrating that ozone concentrations below the current national standards is a threat to the health of even a subset of individuals, the present studies do not provide the evidence to support a change in the 8-hour standards as proposed.

HEALTH AND EXPOSURE ASSESSMENT BRANCH CHIEF BODE: Thank you.

Elizabeth Humphries.

DR. HUMPHRIES: Good morning. My name Dr. Eliza Humphries. I'm a general pediatrician practicing in San Francisco and Marin Counties. And I see many infants and children between the ages of 2 months and 18 years for asthma exacerbations and respiratory illnesses.

I'd like to thank you for the opportunity to talk to you today on behalf of the Northern California American Academy of Pediatrics, which I might add recently published a policy statement on ambient air pollution in December 2004 in piediatrics.

And I'm also here today on behalf of the Health Network for Clean Air, which is a network of health organizations working to improve air quality in California.

I'd like to commend the excellent work done by the California Air Resources Board and the Office of Environmental Health Hazard Assessment in assessing ozone health effects and recommending a new, more stringent health protective standard.

The work that I do in my practice supports the conclusion that people are suffering from ozone pollution and that tighter state standards are necessary if we're truly going to protect everyone from pollution-related illnesses, including infants and children.

As you all know, ozone is a powerful oxidant and respiratory tract irritant. Children and infants are among the most susceptible. They breathe more. They spend more time outdoors. And also their lungs continue to develop and grow through adolescence. In fact 80 percent of the alveoli, or the gas exchanging areas of the lungs, continue to develop postnatally -- or are formed postnatally.

Concerningly, asthma rates have been rising dramatically over the last two decades. And on a personal note, I've seen countless families who bring their children in with asthma exacerbations or wheezing without any family history. The parents are very perplexed as to why their children have now developed the chronic -- the most common chronic disease of childhood. And I found

this also very perplexing and disturbing during my training and decided that I needed to get involved outside of the office.

I think there's ample scientific evidence that ozone and other air pollutants are related to increased asthma exacerbations that cause ER visits, hospitalizations, and missed school days.

Recent studies have also suggested that ozone exposure in children may impair long-term lung function and may not only exacerbate asthma, but actually cause it.

So, in summary, I urge you to adopt the new 8-hour average of 70 parts per billion standard not to be exceeded and the one hour average of 90 parts per billion not to be exceeded. I think adopting the proposed new ozone standards will ensure that the state's air quality goals reflect the most recent scientific research on air quality and health and will ensure that vulnerable populations including children are adequately protected.

Thank you.

HEALTH AND EXPOSURE ASSESSMENT BRANCH CHIEF BODE: Thank you.

Richard Paul.

Do we have a Richard Paul?

Mike Roggee.

MR. ROGGEE: Hello. I'm Mike Roggee with the

California Manufacturers and Technology Association. We represent about 600 of the largest manufacturers and technology companies in the state. And I few remarks that are very general. You won't have to take any notes. And I'll be very brief.

You are the health care professionals. We just want to make sure that you pick standards which are realistic. We implore you to take the time to come up with a decision which is -- you know, take the time to come up with something that can be met. And it concerns the livelihood of hundreds of thousands of Californians.

Between January 2001 and November 2004, 350,000 manufacturing jobs were lost in this state. These proceedings in the draft standards, if implemented, have the potential to create new compliance requirements, which will have a chilling effect on the business growth. It will be difficult to retain companies, let alone entice new companies to move to California.

Since there are no pending deadlines we urge this advisory committee and CARB to take the time to seriously consider the testimony that it heard today and make sure that it gets it right.

Thank you.

HEALTH AND EXPOSURE ASSESSMENT BRANCH CHIEF BODE:

Thank you.

Mr. Roggee, too, I just wanted to remind you too that part of the standard-setting process is not the implementation of controls. And that's all done through a separate process. So --

MR. ROGGEE: I understand that. It has the tendency to promote compliance standards for its --

HEALTH AND EXPOSURE ASSESSMENT BRANCH CHIEF BODE: Great. Okay.

Karen Brunton.

David Schonbrunn.

MR. SCHONBRUNN: Can I go a little bit later?

HEALTH AND EXPOSURE ASSESSMENT BRANCH CHIEF BODE: Sure.

Let's see. Do we have a Sujatha Jahagirdar?

She's on a mailing list. I guess not.

We've got Debbie Shprentz.

MS. SHPRENTZ: Good morning. I'm Deborah Shprentz. I'm a consultant to the National Office of the American Lung Association. And my work focuses on EPA's ongoing review of the national ambient air quality standards for particulate matter and for ozone.

Our message to you today is very simple: Adoption of an 8-hour average California ambient air quality standard for ozone is long overdue and critically needed to protect the health of California residents,

especially the state's infants, children and adolescents.

Ozone is a powerful oxidizing agent that damages lung tissue. Recent research with laboratory animals, clinical subjects and human populations has identified a cascade of adverse health effects from ozone at levels common throughout California. Effects include increased respiratory symptoms, damage to cells of the respiratory tract, pulmonary inflammation, declines in lung function, increased susceptibility to respiratory infections, and increased risk of hospitalization and early death.

Four groups of people are particularly sensitive to ozone: Children, people with chronic obstructive respiratory disease and asthma, people who exercise or work outdoors, and people who for reasons unknown are more sensitive to the physiological effects of ozone, the so called responders. And under California law the air quality standards must be set to protect members of these sensitive population groups with an adequate margin of safety.

The American Lung Association first advocated for the establishment of a separate 6- to 8-hour ozone standard in 1988. And then we had the -- all of the clinical chamber studies of the late eighties to early nineties, which conclusively demonstrated a host of adverse health effects: Decrements in pulmonary function,

increased respiratory symptoms such as cough and shortness of breath, heightened airway responsiveness and inflammation of the airways caused by subchronic exposures to ozone at concentrations below both the EPA and the California 1-hour standards.

When EPA last reviewed the ozone NAAQS in 1996, the American Lung Association supported establishment of a national 8-hour ozone standard at the .07 part per million level to protect against the adverse effects demonstrated in the chamber studies and supported by the epidemiological studies.

And to avoid short-term peak exposures, we favored setting a new 8-hour standard as a supplement to the 1-hour -- the preexisting 1-hour ozone standard.

Thus, the American Lung Association was extremely pleased with the staff recommendations in the draft report to establish a knew 8-hour standard for ozone at .070 parts per million and to retain the 1-hour standard for ozone at .09 parts per million. We feel that both standards are needed to protect all the regions of California from single and multi-hour concentrations of concern, and that that's well demonstrated by the analysis of air quality data in the staff report. It's critically important to retain the 1-hour standard in conjunction with adding the new 8-hour standard.

Now, the health protectiveness of the standards is a function not only of the averaging time in the level, but also of the method used to determine compliance. And we strongly support the not-to-be-exceeded form of the standards and the two significant digits as recommended in the staff report as integral elements of the proposed standards.

In our view the suite of standards proposed in the draft staff report are the minimum necessary to meet the margin of safety requirements of the Children's Environmental Health Protection Act and should not be weakened in any way. We think that the staff of ARB and OEHHA have done a wonderful job summarizing and interpreting literally hundreds of scientific studies on the health hazards of ozone air pollution.

And we agree with their interpretation of the chamber studies on 6.6 to 8-hour exposure, which have reported clinically significant declines in lung function, respiratory symptoms and biochemical evidence of inflammatory damage in healthy young adults at ozone concentrations of .08 parts per million.

Now, as we all know, we can't test babies, young children and those with serious lung disease in the chamber. So these are -- if you look at those results, I think it's obvious that you have to set a standard, an

8-hour standard for at least .070 parts per million to protect the susceptible subgroups which haven't been studied in the chamber studies.

I'd like to point out a recent study that was published in the journal of the American Thoracic Society by Mudway, et al. It came after the staff report came out, so it's not included. But you may want to take a look at it. This was a meta-analysis of 21 human chamber studies where airway responses were assessed using bronchoscopy-base lavage. And they found that linear -- there were linear relationships between ozone dose, airway inflammation and protein leak into the airways over the early and late acute response time periods.

They found that exposure to ozone concentrations at 8-hour concentrations of .08 parts per million at moderate ventilation rates would be sufficient to trigger acute airway inflammation. And the authors noted that since these chamber studies use healthy subjects, individuals with lung disease or other risk factors will experience responses at even lower levels.

I want to mention briefly several other studies that have come out since the publication of the staff report dealing with ozone and acute mortality. There's been a lot of discussion of the 14-year 95-city NMMAPS study. But I think what hasn't been mentioned is that the

relationship between mortality and ozone reported in the study was evident even on days when pollution levels were below the 8-hour average level of .08, and that these results did not appear to be confounded by temperature or PM10.

Secondly, there was a large multi-city European study, the APHEA2 study, which also reported a positive association between both 1- and 8-hour concentrations of ozone and daily mortality, particularly respiratory mortality during the warm season.

And, third, there was a recent case crossover study of 14 U.S. cities that concluded that the associations reported in these other studies between ozone and mortality risk are unlikely to be due to confounding by temperature.

Just the point I would make about these studies is I think that they emphasize the appropriateness of including mortality as a health endpoint in the benefits assessment, as has been done in the draft paper. And we'd just like to note that the methodology used here tracks closely that used by EPA to estimate ozone -- benefits of ozone control and that this methodology has been reviewed and approved by the independent EPA Science Advisory Board.

Now, of course there are many other lines of

evidence that support the proposed standards. The animal toxicology studies showed that long-term exposures to ozone can result in remodeling of the airways of the lungs and deposition of collagen. And since the completion of EPA's review of the standards in 1996 there have been a number of important epidemiological studies on the long-term impacts of ozone exposure on lung function. And the draft report reviews these studies of seasonal and multi-year exposure to ozone and particularly those that show that these exposures may be related to the development of asthma among children active in several sports.

There are some European studies showing a link between ozone and diminished lung-function growth, and that these studies further support the proposed 8-hour standards, because in fact attainment of a combination of the 1-hour and 8-hour standards will lower the long-term year-round concentrations of ozone in California.

In conclusion, we believe that the policy recommendations in the staff report are fully supported by the scientific evidence, and we urge this Committee to fully endorse the recommendations and urge their adoption by the California Air Resources Board.

Thank you.

HEALTH AND EXPOSURE ASSESSMENT BRANCH CHIEF BODE:

Thank you.

Sonya Lunder.

MS. LUNDER: Hello. My name is Sonya Lunder. I'm an environmental analyst with the Advocacy Organization Environmental Working Group. Our mission is to assure that the public's health is fully protected from environmental chemical exposures in our food, water and air.

Environmental Working Group strongly supports the efforts of ARB scientists and the expert review panel to define an ozone standard that is fully protective of the health of Californians.

I was very impressed by the materials prepared by the Air Resources Board in defense of the proposed standards and awed by the estimated health burden attributed to reductions in ozone pollution.

I wasn't intending to make comments at this meeting. However, yesterday's panel discussions touched upon findings of respiratory effects in animals experimental studies at levels at or below 0.07 parts per million. This piqued my concerns about the ability of the proposed standard to fully protect children, people with respiratory illnesses and other vulnerable groups from ozone pollution.

In experimental or chamber studies it appears

that some individuals are consistently affected by ozone levels at or near the proposed standard of 70 parts per billion. Even effects on a small number of study participants, for example, 10 percent might be labeled as a responders, will translate into a significant health impact when we can consider exposure to millions of Californians. If this is indeed the case, the proposed standards may provide inadequate protection, especially in light of the regulatory objective defined as determining the highest pollutant concentration for a given time that is unlikely to induce adverse effects in any one who's exposed.

My second concern relates to the children and other vulnerable subgroups. These groups are not -- with compromised health are not able to participate in chamber studies, and there are significant data gaps including the vulnerability of the developing fetus, the sensitivity of these population to really low level of concentrations of ozone and the effect of ozone and its ability to alter -- permanently alter lung development or trigger new cases of asthma.

Given that there's little or no margin of safety between the proposed standard and effects in healthy adults, our concern is that vulnerable groups may be more at risk when exposed to ozone concentrations of 70 parts

per billion.

Our group's concerns could be assuaged by the assurance by the review committee and by air district staff that individuals and controls in epidemiological study are not indeed responding at this concentration of 70 parts per billion over long-term exposures or that the data gaps in children's respiratory health including their lung physiology and fetal exposures do not in your approximation raise concerns that children might be more affected at these levels; and/or if it's impossible for you as expert scientists to give us that assurance, we would promote the addition of an additional safety factor. And we think that would be warranted to guaranty that the new 8-hour standard achieves its intended goal.

Thank you very much.

HEALTH AND EXPOSURE ASSESSMENT BRANCH CHIEF BODE:

Okay. I've got -- actually I've got three more people.

David Schonbrunn, ready?

MR. SCHONBRUNN: Good morning. David Schonbrunn, President of TranSDEF, the Transportation Solutions Defense and Education Fund.

We are active in the San Francisco Bay Area pushing regulatory agencies to enforce existing air quality standards. We are in the trenches with the

products of your work.

Our organization strongly supports the proposed standard and especially appreciates the form of the standard, with three decimal places. Our colleagues are in federal court challenging the rounding protocol in the federal 1-hour standard. We urge you to revisit the precision of the state 1-hour standard and move it to three decimal places, as something that would be very beneficial in establishing clarity in terms of rounding protocols.

In reviewing the comments on the recommendation I detected a significant skewing of the data. All the comments from humans supported the recommended standard. The only opposition to the recommended standard was from industry.

May I remind you that the industrial corporation is an opportunistic life form that, unless checked, destroys other species and habitats. They have no interest in the health of humans.

Rather than setting standards based on the convenience or profits of industry, I am very proud that our state has adopted a standard to protect the most sensitive humans. I'm very pleased that the Legislature chose that standard and today's recommendation is designed to accomplish that.

What we're talking about here is partly precautionary principle. That hasn't been mentioned yet, and I think it does need to be mentioned. Industry has produced no evidence that humans suffer due to an inadequate level of ozone. Reducing exposure to ozone is a good thing for human health. The proposal is responsible and prudent. And it certainly is better than the existing standard. So on that basis we urge you to support it.

If we step back for a moment from the specific objections raised in the earlier public testimony, we realize that industry never concludes that enough research has been done or that data are consistent enough to regulate them. On the other hand, the perchlorate study that just came out of the National Academy of Sciences -- I think that was yesterday -- is considered definitive. Funny how the study relieves the rocket industry of billions of dollars of liability. This is a cynical manipulation of the scientific process. Please place the objections that you heard this morning from industry in just that context. Industry's interests are antithetical to human health. Just stop for a moment and remember the history of the auto industry rejecting seat belts, rejecting air bags, having Pintos and things like that.

I personally am pleased by the work of ARB and

OEHHA, as a former asthmatic myself and with a series of family members with COPD. I feel you are working on my behalf. Thank you very much for your excellent work.

HEALTH AND EXPOSURE ASSESSMENT BRANCH CHIEF BODE: Okay. I've got actually two people left, Curtis Moore and Henry Gong.

Was there anyone else besides that?

Then Curtis Moore.

MR. MOORE: Hi. My name is Curtis Moore, and I'm not representing an organization. Indeed, I'm not even representing myself.

(Laughter.)

MR. MOORE: I'm the co-editor and publisher of the Health and Clean Air Newsletter. I have some copies of it over on the table and some CD's. This is a newsletter that reviews scientific literature, scientific studies as they enter the literature and tries to explain them in layman's terms.

My co-editor and close personal friend, Dr. David Bates, is a retired Dean of the School of Medicine at the University of British Columbia and a Professor Emeritus there, a member of the National Academy of Sciences, and some would say the best environmental epidemiologist in North America.

David and I wanted to bring to your attention

that in press currently are three studies relating to ozone and mortality. These are in the journal Epidemiology. And David was invited to write an editorial on these studies. And David, who lives in Vancouver, British Columbia, is unable to travel. And he asked that I share his editorial with you because it does summarize these studies. I'm sorry. We attempted to obtain pre-publication copies of them, but were unable to do so. Nevertheless this is a -- as usual from David, an excellent summary of these. I'll just read his editorial, because that's the simplest way to do it.

And since I'm merely a conduit here, it would be hopeless to ask me any questions.

(Laughter.)

MR. MOORE: "This issue of Epidemiology contains three meta-analyses of the extensive data relating ambient ozone levels to daily mortality. When I was a student at Cambridge my tutor used to throw things across the table at us if we did not always mention the most important fact first. So for those whose attention span is dwindling, all three studies report a significant association between

ozone levels and total mortality.

"These studies were commissioned by the same agency, but the authors were free to carry out the analysis as they saw fit, and all three differ.

"One author, Levy, used data from 14 U.S. cities, 13 Canadian cities, and 21 European cities, and excluded data from Mexico City and the National Morbidity and Mortality Air Pollution Study or NMMAPS.

The second study, Bell, used the data from the NMMAPS study of 95 cities, together with European studies, for a total input of 144 data sets. These authors had already published an analysis of the NMMAPS data" -- which you've been discussing this morning -- "alone.

"The third, Ito, was more restricted and used data from 7 U.S. cities plus other worldwide data for different parts of the analysis.

"Another difference was that one author, Levy, used data on the

prevalence of airconditioning in both the United States and Canada.

"Bayesian hierarchical models were used in the analyses. Particulate matter interaction with ozone was found generally to be unimportant.

"All three studies noted that the response function was higher in the summer, when ozone levels are higher, than in the winter. And this means that if the data are not stratified by season, the overall response income is likely to be diminished.

"Other factors noted were that the prevalence of airconditioning affected the outcome (Levy), that the NMMAPS data alone yielded lower response outcomes than most other analyses, and that there was a generally satisfactory concordance between U.S. and European data.

"One study found a change in total mortality of 0.86 percent per 10 parts per billion in summer (Levy); the second (Bell) found the change of 0.83 percent per 10 ppb and total mortality overall

and agreed that the U.S. and non-U.S. data were similar; and the third study (Ito) provided a detailed seasonally background and showed that the main effect occurred in the warm season.

"In an analysis for a single pollutant model, data from 8 U.S. regions, 8 European cities, 2 Australian cities, plus Mexico City, Sao Paulo, Santiago, and 2 regions of South Korea are plotted in the Ito paper. And below zero data, or insignificant, were noted for 5 cities. All the rest were positive. The highest was for Brisbane in Australia at about 3.5 percent mortality for 10 ppb for the 24-hour average ozone.

"Reviewing all the data, I would regard the value of 0.86 percent change in mortality per 10 ppb as a minimal figure since inclusion of data from Brisbane and Mexico City would increase this significantly.

"The European data was derived from 23 different regions with mortality data

over a 3-year period. The authors reported no association between ozone and mortality over the winter months, but a significant association in summer, with a mean increase of 0.33 percent in total mortality, 0.45 percent in cardiovascular deaths, and 1.13 percent in respiratory deaths, for an increase of 10 micrograms per cubic meter of ozone. As 10 micrograms is equivalent to 5 parts per billion, the percentage increases should all be doubled for a 10 ppb change." Thus -- and these are my words -- the increases would be 0.66 percent in total mortality, 0.90 percent in cardiovascular deaths, and 2.2 percent in respiratory deaths.

They also found that PM10 values were not a confounder but reported some possible interaction with NO and CO.

"We have known since Haagen-Smit's work in 1952 that tropospheric zone is formed from nitrogen dioxide and the presence of hydrocarbons and sunlight in a complex series of reactions. It is a

difficult pollutant to control owing to the complex nature of its formulation.

"But there are other aspects that make epidemiological studies difficult. And the northern hemisphere cities have a distinct ozone season, and this includes Los Angeles.

"The correlations between temperature and ozone levels are high and this makes analyses complex because heat waves are themselves responsible for an increase in mortality. It has recently been calculated, for example, that in the Netherlands in the recent severe heat wave over 400 deaths originally attributed to the heat were probably due to the accompanying high ozone levels. The same effect was present in Britain. The interrelationship between heat and tropospheric ozone is not straightforward.

"Atmospheric scientists in Toronto have recently shown not only that surface temperature and ambient ozone

are related, but published evidence that elevated ozone levels have the effect of increasing surface temperatures.

"In Brisbane in Australia and in Mexico city levels of ozone varied little throughout the year. Hence there is no seasonality factor. It may be therefore significant that it is these two cities that yield the highest response outcome, as they are the least likely to be confounded by other seasonality factors. If this is the case, excluding them from a meta-analysis will necessarily have the effect of lowering the dose response metric.

"Secondly, the question of personal exposures is also unfortunately complex. This comes about because of the many factors that contribute to the formation of ozone and because emitted NO from vehicles scavenges ozone to form NO2. Hence values in the center of heavily urbanized cities will be lower than in the suburbs. This might mean that the

most vulnerable members of a population are personally exposed to lower levels than are wealthier people in the suburbs.

"One of the studies (Levy) took the prevalence of airconditioning into account. Homes with such units will have lower indoor ozone levels than those without. Another factor is introduced by the time course of tropospheric ozone formation, which usually reaches a maximum between noon and 3 p.m. Children coming out of a school where, if air conditioned, the indoor ozone level will only be about 15 percent of ambient, will thus encounter the highest ozone level of the day, and this just at a time when physical exercise out of doors will be commoner. While it is possible to list these factors which may account for individual differences in ozone exposure, it is not easy to incorporate them in any meaningful way into the studies of outcomes from ozone exposure.

"Thirdly, in northern latitudes all respiratory illnesses and, hence, all outcomes such as hospital admissions and respiratory mortality are at their highest during winter months when ambient ozone is at its lowest. It has taken some years for the impact of this to sink in. But it was for this reason that in my analysis of southern Ontario data in a time series study in 1987 I stratified the data before the analysis into summer and winter seasons and found a highly significant relationship between ambient ozone and hospital respiratory admissions, but only in the summer. If summer and winter data are combined in one analysis, it may be difficult to see any ozone effect at all due to the fact that respiratory illnesses are the commonest in the winter months when ambient ozone is at its lowest.

"Nevertheless a research group in New Jersey has recently concluded that the association between ambient ozone

levels and hospital emergency and admission data for asthma are so consistent in that state that ambient ozone levels in the summer can reliably be predicted from the health data. They reported sufficient databases exist for emergency room visits by asthmatics in the northern central New Jersey and throughout the state for hospital admissions for these health outcomes to be used as health based indicators, complementing air monitoring data in assessing whether improvements in public health are occurring because of reductions in emissions of precursors of ozone.

"These three new meta-analyses and the European study, each with unique features, appears to resolve the question of whether ambient ozone levels are associated with increased mortality.

"It seems unlikely that PM2.5 is an important confounder and the effect appears to be independent of temperature.

"A final question, biological plausibility, is in some ways the easiest to answer. Ozone is capable of causing inflammation in the lung at lower concentrations than any other gas. Such an induced effect would be a hazard to anyone with heart failure and pulmonary congestion and would worsen the functioning condition of anyone with advanced lung disease. The ozone mortality relationship is therefore supported by strong biological plausibility.

"Those who follow ozone closely will have noticed that recent data indicate that, 'background'" -- which of course is not the same as natural background -- "levels have been steadily rising in both hemispheres and that increasing emission of precursors in Asia, particularly NO2, are predicted to increase the background ozone level in western America and Canada by between 5 and 10 ppb. Levels of ozone over the Atlantic Ocean have also been rising.

Global warming is also expected to increase tropospheric ozone levels, though the magnitude of this effect is uncertain. Ozone is simply no respecter of frontiers.

"Canada and the World Health Organization have both proposed a standard or guideline of .06 ppm for an 8-hour exposure. The United Kingdom is aiming at a .05 ppm for 8 hour as a maximum by the year 2005. U.S. EPA will be reviewing the federal ozone standard during 2005. All these jurisdictions recognize that what is involved is a delicate balancing act, with no margin between present exposure levels and adverse effects on health.

"In Los Angeles ozone exposures in school children have been shown to be associated with school absences for respiratory illnesses. And the economists have been busy calculating what these represent in terms of economic burden. When one adds together such effects on a national basis, plus

the influence of ozone on hospital admissions and emergency visits, and now the impact of premature mortality, which would have to be included, one does not have to be an economist to see that the overall economic burden of this pollutant must be enormous.

"In such a context no one could maintain that these three meta-analyses, which support European data, are of academic interest only. What they point to is the urgent need to reduce public exposures to ambient ozone by all possible means."

Again, this was a personal statement by David Bates, not in his capacity as a co-editor of the newsletter. But given his level of expertise, he thought you might be interested in these.

HEALTH AND EXPOSURE ASSESSMENT BRANCH CHIEF BODE: Henry Gong.

BOARD MEMBER GONG: I'm the only member of the Air Resources Board to be at this AQAC meeting fortunately. And I'd like to take this opportunity to say that as a representative of the Board that I thank Dr. Kleinman and his esteemed committee for an excellent job

of evaluating the scientific basis for the state ozone standard.

AQAC had an excellent yesterday, an open and candid discussion that brought forth important insights about both major and minor issues. As with many comprehensive scientific reviews of this nature, much behind-the-scene writing and deliberation occurs. For that, the OEHHA staff and ARB staff and the chapter authors, some of whom are still sitting in the audience, wondering when they'll be called, are to be commended for their tireless and thoughtful commitment to this important task.

In addition, the subsequent comments and responses by staff were very revealing and useful in that the presented issues I think really help everyone, including the Board, to better define, refine, focus and understand what points are uncertain and can be clarified or improved.

And indeed I'm looking forward to a subsequent discussion from staff regarding some of the comments presented this morning from the commenters.

I also found it hard to resist, but as a former AQAC member and after hearing yesterday's discussion I was actually stimulated to ask the following technical or scientific question to AQAC about adverse effects.

Some of you -- let me get my right line here. Some of you may have heard a variation of this theme before. But I was wondering, if there was time, could AQAC members briefly comment about the selection of endpoints or health effects from ozone exposure.

Dr. Delfino already mentioned about mortality as an endpoint this morning, that it was sort of a gross or late endpoint to look at, or health outcome, although it is very important.

This issue may also depend on the types of subjects you're interested in. Nonetheless, there are endpoints and there are endpoints. Symptoms, as was pointed out, tend to be less reported in exposed children than in adults. We don't understand this phenomenon.

Some endpoints, such as barometry and FEV1, may show significant decrements in up to 25 percent of the exposed group. In fact, the FEV1 response has poor correlation to symptoms in airway inflammation.

Some endpoints may be very sensitive, on the other hand, such as inflammatory markers. And examples of this are exhaled nitric oxide and sputum neutrophils or eosinophils.

In fact, these types of markers can precede or perhaps even predict respiratory exacerbations certainly in asthmatics and are being used to make patient care

decisions in some clinics.

The timing of the measurement of endpoints is also a critical decision since some changes such as sputum neutrophilia may peak at different post-exposure times than immunologic or biochemical changes.

The response to ozone is a complex dynamic process, to be sure, depending on what effect you select to measure. So the pregnant questions I have are:

Is there a sensitive adverse effect that we should be looking for?

Is there a best adverse effect? -- you can say that -- or a combination of adverse effects for us to follow and look for?

With that I'd like to conclude my comments and, again, thank the AQAC for an excellent job. The Board looks forward to your advice. And thank you again.

HEALTH AND EXPOSURE ASSESSMENT BRANCH CHIEF BODE:

Thank you.

That concludes are oral public comments.

And, Dr. Kleinman, then if you'd like, we can go right into staff summary of written comments and responses to comments.

CHAIRPERSON KLEINMAN: Okay. However, before we do that, I just wanted to see if anybody on the Committee wanted to accept Dr. Gong's challenge to discuss potential

endpoints.

ADVISORY COMMITTEE MEMBER DELFINO: Just a point of clarification on my statement about mortality.

I was mostly referring to total mortality, that it's a rather blunt outcome and that from a clinical standpoint we're always more interested in at least specific types of outcomes like cardiovascular and respiratory in the very least. And I think the statement from the colleague of Dr. Bates is very important in that these new analyses, in addition to the NMMAPS and other existing analyses, looking at specific outcomes are quite informative in showing the strongest associations for respiratory outcomes. And of course kids with asthma don't usually die, and so mortality from asthma is irrelevant. And so, in fact, morbidity from asthma is the key outcome. So it really depends on the outcome, just as a point of clarification.

CHAIRPERSON KLEINMAN: The other issue from my own research, the timing of measuring various endpoints is a critical factor, as Dr. Gong mentions. And our burgeoning understanding of the signaling characteristics of the irritation and inflammation process is getting to the point where we may be able to start to identify a suite of potential endpoints that could be followed.

And, unfortunately, they don't all occur at the

same time, which makes it very difficult to study under controlled conditions with humans or in an epidemiological setting. And really until we have completed more mechanistic studies, which I think are essential, we are going to be limited in our ability to pick endpoints that seem to respond sensitively to provocation by various pollutants, ozone certainly being one of the most prevalent for chamber studies.

So I agree that this is an area that needs a great deal more of research. But I think that the information that's been acquired to date has been very good, very useful; and to some extent we may be able to go back and reevaluate it in the light of some of the new understandings of the timing of various responses, to partially get a picture of why some studies seem to see a response given the same exposure, others do not.

And it may be partially in terms of the exposure protocol, the measurement protocol and things like that do need to be carefully considered in comparing results from one study to another.

I'd like to go ahead and move directly into the staff responses at this point to the oral presentations and also to the written presentations that were provided at the public review period.

HEALTH AND EXPOSURE ASSESSMENT BRANCH CHIEF BODE:

Thank you, Dr. Kleinman. I'm going to have Dr. Deborah Drechsler, who's the lead person for the Air Resources Board on the staff review, begin discussing comments and responses.

DR. DRECHSLER: Okay. I need my slides.

(Thereupon an overhead presentation was Presented as follows.)

DR. DRECHSLER: Okay. We'll try that. This is microphone seems to have a mind of its own.

I'm going to give you a part of the presentation of the staff responses to the public comments. And Dr. Ostro from OEHHA will also present part of this.

One of our ARB staff people, Larry Larson, will also be coming up during the discussion on background ozone.

--o0o--

DR. DRECHSLER: The public comments fell into six major categories:

The method and process used for standard review;

The form and attainability of the proposed standards;

Natural background concentration;

The adequacy of the scientific evidence supporting the recommendations;

Justification for the recommendations; and

That the health benefits analysis is flawed.

--o0o--

DR. DRECHSLER: Several commenters recommended that OEHHA and ARB follow the federal process for ambient air quality standard review. Since the action before us is a state regulation, the federal process and procedures do not apply. The California Legislature passed a law some time ago called the Administrative Procedure Act which outlines the steps and requirements for adoption of state regulations, and we have followed this procedure in our proceedings.

They also -- there were also several comments that we did not perform a risk assessment as part of the standard review. And state law requires that standards be health based.

The Legislature defined an air quality standard in the Health and Safety Code as an exposure which includes an averaging time and a concentration; and, in effect, defines a standard as a maximum exposure that is estimated to be without effect in anyone who undergoes that exposure.

Since the standards are essentially a safe maximum exposure, risk assessment and probability of experiencing those exposures are not really relevant to the proceedings. And these comments seemed to be based on

a misunderstanding of the definition of a standard under California law.

And this also points to the central question that OEHHA and ARB addressed in our review, that based on this definition of an ambient air quality standard, the key question is: What is a maximum safe exposure? And that's a point that I'd like to emphasize for the Committee to consider when they make their determinations on the adequacy of our recommendations.

--o0o--

DR. DRECHSLER: Several commenters also recommended that the federal form allowing several exceedances per year be adopted instead of the not-to-be-exceeded form that we have proposed. Historically, California has used the not-to-be-exceeded form, and this also comes out of the definition of the standard as a maximum safe exposure. California has historically used this form, and as a state regulation the federal form is not required.

Furthermore, this form is supported by the scientific evidence that we have used to support the recommendation and specifically studies that suggest that multiple acute exposures can over time lead to morphological damage to the lung tissues that accumulates.

--o0o--

DR. DRECHSLER: Several commenters also raised the issue of the attainment designation process and recommended that it be changed. One commenter also sent us a proposed method to replace the current one.

Currently California uses the expected peak day or EPDC method for determining attainment designations. And this is set in the California Code of Regulations under sections that are not related to those we have opened in this proceeding. It's a completely separate process.

The California standards are health based and attainability is not a consideration under the state law. Whether or not a standard can be attained is related to the attainment designation process.

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DR. DRECHSLER: We also received a number of comments related to background ozone. And several of the speakers this morning went over their written comments and added to them somewhat.

ARB used the range of .03 to .05 with a mean value of .04 parts per million to characterize background ozone in California.

Several commenters asserted that the natural background is higher than this and that the proposed standards overlap background.

ARB does not agree with the analyses presented by the commenters. And we will have several of our staff address that in more detail.

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DR. DRECHSLER: We also received a comment that tropopause folding events, also known as stratospheric ozone intrusions, will lead to exceedances of the standards.

Historically there has been only one documented tropopause folding event in California. This occurred in 1972 in Santa Rosa. And based on this we do not think that this is going to be a common cause for ozone standard exceedances.

In addition, policies and procedures are already in place to handle such events if any are demonstrated to occur in the future. And --

HEALTH AND EXPOSURE ASSESSMENT BRANCH CHIEF BODE:

Dr. Kleinman, this is a point where we've got Larry Larson from our Planning and Technical Support Division, who's done a little analysis in some of this background in stress -- and intrusion type issues. And I wondered if you -- did want to hear more detail on that? I think yesterday you mentioned you wanted to hear some detail on the analysis.

CHAIRPERSON KLEINMAN: I think it's worthwhile.

HEALTH AND EXPOSURE ASSESSMENT BRANCH CHIEF BODE: Okay. Then we'll have Larry break in right now.

MR. LARSEN: I don't trust computers. Can I have the overhead?

(Laughter.)

MR. LARSEN: The presentation won't be a particularly long one. Also, it's not meant to really address every potential issue.

We've clearly been looking at some of the import of the comments concerning background that came in. And the material in chapter 4 in our staff report lays a lot of very good background.

(Laughter.)

(Thereupon an overhead presentation was Presented as follows.)

MR. LARSEN: As you can tell, this is a very polished presentation.

(Laughter.)

MR. LARSEN: We talk about policy-relevant background, and it's been mentioned here today too. I just want to say briefly that we've noted that policy has some various portions to it and there are policies that guide the establishment of a standard and there are policies that guide planning for attainment and emission reductions, all those things. We said that background

itself is not directly relevant to the policies that guide setting the standard. It's interesting, but it's not -- it's at a tangent to the issue of establishing a concentration and duration and monitoring method and pollutant. So it really goes over into the other arena that we're not talking about here today, which is: When a standard is established, what do you do to attain it? That's really where a lot of the comments come in.

So it becomes relevant really in the planning process outside of the venue when we talk about what policies it's relevant to.

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MR. LARSEN: So the staff report in Chapter 4 indicates actually a range. You've heard that the staff report says that 40 is the maximum. We really didn't say that so explicitly. Or if we did in one place, it's conflicted with another place where we really indicate a range.

Wrong slide. I have to come back to that one.

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MR. LARSEN: And here's the basic issue. I have two little scenarios, scenario A and scenario B. Consider two different days and consider these are the highest ozone days of the year.

In scenario A the green portion at the bottom is

below 40 parts per billion, which I'm going to say let's use that for this example as background.

Then the green part is background the red part up above the line in scenario A would be from human origin, human influence. And if we can reduce that human influence by half, then we would take a value down from 80 ppb, from 80 ppb here down to -- in the future cutting the 40 -- above 40 background in half, we'd be down to 60. And we would bring that day down below the level that you're considering for the standard.

So the concern in a lot of people's minds about the attainability of a standard is really more scenario B, where you have only a 10 parts per billion due to human influence and a 70 parts per billion due to background all by itself. So that in the future if we cut the 10 in half, we'd reduce that to 5. But you added onto the 70 background and you don't attain.

And that's the kind of question people have been raising: Does background actually get up into this 70 ppb range where human activity would add on top of it? And that's the general issue, just to lay that before you.

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MR. LARSEN: Stratospheric ozone is one of the natural background parts that's been talked about. And I think that's really where some of the interest lies, as

people have spoken to us.

Stratospheric has two parts to it. There's the big potential, these big stratospheric ozone intrusions associated with tropopause folding events. And that can bring large concentrations down to the surface. Usually though it doesn't go down very far to the surface. What -- I consider this line right here to be about 30,000 feet. So down here at 3,000 feet and below would be really where most of the people are, and it gets fewer and fewer people as you go up to that high level. So most of the time these big intrusions don't penetrate down all that far. Only rarely do they get way down here.

The other example is really small amounts that are chronic, like chronic leakage of stratospheric ozone downward. And we call that due to stratospheric general subsidence going downward in the atmosphere. But that does not really introduce large amounts down to the surface. It's small amounts, and the staff report addresses that as well.

So just trying to keep all the pieces of the puzzle in place, it's a challenge to assemble all of those into a coherent picture.

A key part here is that analysis of ground level ozone data that we've been looking at is, in my opinion -- that's what IMO means there -- in my opinion, is very

unlikely to be able to tease apart 5 parts per billion, 10, perhaps 20 on occasion, that might be attributable to general subsidence or general leakage downward. That would be part of tease apart.

But when it comes to the big events like this, I think we have much more prospect of being able to identify such things and take them into consideration as outside the control of emission reduction requirements, which is part of our planning process. And you heard from Deborah that we have policies and procedures in place to be able to grapple with such odd and extreme events.

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MR. LARSEN: It's worth I think showing you this, because I think you'd have an interest in it and not fall asleep.

We say that ozone transport from the stratosphere down to low levels like coastal-populated levels, the Central Valley in California, those places, are really quite uncommon, it seems. Now, we said we only know of one documented case -- and I say "question mark" because I'm not sure I had an absolute comprehensive grasp of literature -- but documented back in November '72 at Santa Rosa in the San Francisco Bay Area. It involved intense down drafts due to low pressure system accompanied with heavy rain. That's not usually the kind of day we think

of as our design days for ozone attainment. So the six coastal -- oops. Forget that last bullet since I'm not going there.

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MR. LARSEN: But here's what that day looked like. This is the ozone data from that day. You have the 16th, 17th, 18th -- the 19th of November looked like this. This spike is way, way above anything you expect in November at Santa Rosa. And it can have that kind of a spike associated with it. And it just stands out and catches your eye, and you say, "What went wrong? Was somebody xeroxing things under the probe and making" -- never mind.

(Laughter.)

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MR. LARSEN: We have been looking into incidents of very low ozone, right around the level of the standard you're considering, in Coastal areas just to see where they might come from, whether we think that there are incidents in which 70 ppb ozone is occurring simply from background or not.

But here is a backward trajectory, a -- following the air parcel backward from that tropopause folding event in November 19th, 1972, where did that air come from? It came from way out over the ocean. This is a 72-hour back

trajectory. So it's not a -- we wouldn't say that's very much associated with human involvement. But it reached that 180 parts per billion for one hour in November 19th from the stratospheric drastic down-wash.

I want to contrast this trajectory with other trajectories, which is why I showed you.

On the other hand, when we look at something along the coast at a place like Vandenberg Air Force Base, where you had I believe on this day a 77 parts per billion maximum 8-hour average at Vandenberg Air Force Base on the coast, we normally think of that as a nice coastal clean ocean site. Doesn't have a lot of high ozone days. But this day it did. And when you follow the back trajectories, it takes you right over San Joaquin Valley. Here's Bakersfield; here's Fresno.

In other words, when we back the air back through a populated area like that, known to be a source of lots of emissions, we would not assume that that represents background ozone. We'd have to assume there's a substantial human input to that process, which would respond to controls and would bring us down from 77 ppb down below the proposed standard that you're looking at.

This day was either the highest or second highest day at Vandenberg. And all of the high days at Vandenberg that we looked at tracked backwards over to populated

areas. In other words they don't look like background ozone to us.

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MR. LARSEN: Now, one of your comments came from Dr. Lefohn pointing out this data at Vandenberg. I want to be clear that he was not proposing that this 77 ppb represented background. His purpose in presenting this one was that it was the spring time or the late fall, not the middle of summer. And our ozone season can have some relatively high values in some places. The seasonality doesn't always have high in the middle of summer and low in the spring and fall. So all of our coastal areas tend to have a spring and fall high ozone season.

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MR. LARSEN: And the last thing I want to touch on concerns the point that was made about the benefits analysis. And it -- it's very quick. And the point was taken that if we used the 40 ppb background, we applied that to all days -- and the idea in the benefit stuff was here's vertical line at 40 ppb, and we're looking at reducing the shaded part downward, sandwiching it between the standard you're proposing at 40 ppb, or whatever background is -- and we applied 40 ppb to all days because we don't know how to handle day by day by day how much was background and how much is human, to be able to tease that

apart is beyond our -- the richness of the data sets that we have available to work with.

So what we really used was the 30 to 50 ppb as a range in our thinking. But we don't know how to apply the range to this day by day generic rollback. So the rollback procedure we used in here applied a general 40 really to give you an estimate, to ballpark those benefits in a way that seemed reasonable.

It would be nice to be able to do it in more detail. We're preparing more detailed analyses and some explanations for stuff for the staff report and for comments. But it's a work in progress, and the handwritten slides you see here really demonstrate that it's a work in progress.

DR. DRECHSLER: Okay. The next category of comments dealt with the adequacy of the scientific evidence. And there were three main issues under this topic:

First, the definition of an adverse health effect.

And then several issues were raised with reference to the controlled human studies.

And there were also comments made that the epidemiologic studies were flawed.

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DR. DRECHSLER: Several commenters presented the view that the acute effects that we attributed to ozone exposure do not meet the standards of being judged adverse.

The American Thoracic Society published guidelines for assessing adverse effects of air pollution exposure in 1985 and 2000. And we used both of these guidelines in evaluating whether or not we considered an effect to be adverse.

In general, in terms of acute studies, an effect was considered significant if it was large enough to reduce or limit work or exercise capacity or was sufficient to impact quality of life. Epidemiologic studies investigated a number of endpoints which would clearly qualify as adverse such as hospitalization, emergency room visits, mortality, and school and work loss days.

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DR. DRECHSLER: In terms of controlled exposure studies, the comment was made that the protocols used were unrealistic to the general population.

And these protocols have been pretty much standardized over the past about 30 years. They were originally designed to simulate real-world activity patterns of people likely to have the greatest outdoor

ozone exposure, which would include children, recreational athletes, outdoor workers, people doing home maintenance and yard work and those sorts of activities.

The continuous exercise 1-hour protocol is representative of children playing, personal exercise programs and relatively short periods of outdoor home and yard maintenance.

The 2-hour intermittent exercise protocol is representative of slightly longer term, more intermittent outdoor activity, which would include some sorts of sports and recreation activities, exercise programs and other home and work-related activities.

The 6.6- to 8-hour protocols were designed to simulate a full day of outdoor work.

And the exercise rates used in these studies are based on actual measurements of people who were performing representative activities for those exposure times. And, thus, we believe that the exposure patterns in these studies are relevant to assessing responses to people who would undergo similar patterns of real-world exposure.

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DR. DRECHSLER: There was also an issue raised about possible subject response bias. One commenter believed that -- apparently believed that subjects might, if you'll excuse the somewhat colloquial word, fake

responses because they smell ozone in the chamber on their entry.

This was fairly unlikely because, first, ozone dulls the sense of smell quite quickly. And also some investigators to preclude this possibility introduce a trace of ozone in the chamber on the subject's entry, which is gone within a couple minutes of the beginning of the exposure. But the sense of smell has been deadened during that time period. So the time period during which the subject actually notices the ozone odor is about the same in the ozone compared to the non-ozone condition.

Studies that have not introduced the trace of ozone during the filtered air exposure have come up with results that are comparable to those that did.

In the case of pulmonary function tests one of the commenters today indicated that these tests are very effort dependent. There is an element of that. But the standard protocol in these types of studies is to follow the American Thoracic Society guidelines for acceptability and reproducibility of lung function tests. And it's customary standard practice for the subject to perform at least three tests maneuvers and that they must agree within 5 percent, which is the ATS criteria.

It's very obvious looking at the tracings of these sorts of tests whether or not the subject is making

a maximal effort.

And one of the reasons that FEV1 is frequently used as the major measurement from pulmonary function tests is that it is relatively effort independent and it also has the lowest coefficient of variation between repeated tests. It's basically impossible for a person to perform consistent lung function tests unless they're making a maximal effort.

In addition, subjects have no control over their responses to airway bronchio-challenges or inflammatory endpoints. And at least with certain symptoms, particularly coughing, it's obvious to the observer whether or not the subject is experiencing this.

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DR. DRECHSLER: Another comment related to controlled exposure studies was that responses to ozone conditions should be compared to a background ozone concentration, for example, .04 parts per million rather than to filtered air. This apparently is based on the view that response is related to the change in ozone concentration. And this is a misunderstanding of the dose response relationship with ozone exposure. Ozone effects are related to the total inhaled dose and to the dose rate, not to incremental change in concentration.

The ventilation rate and the duration of exposure

also factor into responses. And on an individual level the dose response relationship is not really a linear function. On an individual level, there -- we can identify a threshold -- or what you could call a threshold concentration. But because of the variability among individual people, it's difficult to identify threshold on a population level.

Further, although there's only one study available that investigated responses at .04 parts per million, the study did not find responses at that level. And it's unlikely that if we compared -- had a larger number of studies and data points at that concentration, the results would be different using .04 as the baseline compared to filtered air at the baseline.

And now I'm going to turn this over to Bart Ostro from OEHHA who's going to discuss the comments related to epidemiology and to benefits analysis and the justification for the recommendations.

OEHHA AIR TOXICOLOGY AND EPIDEMIOLOGY SECTION

SUPERVISOR OSTRO: In the record of some epidemiologic studies and the studies used in the benefit analysis there was a series of comments indicating concern that the statistical modeling issues were not fully acknowledged.

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OEHHA AIR TOXICOLOGY AND EPIDEMIOLOGY SECTION

SUPERVISOR OSTRO: Our response was, and is, that most of the modeling issues were discussed for each type of epidemiologic study. And the Committee advised yesterday that we maybe move that discussion up in our document and make it a little bit more clearer about which endpoints are covered.

There were some additional issues and uncertainties that were cited in the comments that we will in fact add to our document, some other points there. So those points are noted and we will add those.

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OEHHA AIR TOXICOLOGY AND EPIDEMIOLOGY SECTION

SUPERVISOR OSTRO: A second issue that was common to many of the comments was that there are inconsistent results between studies and variability in the results.

So our responses are that heterogeneity was acknowledged in the report, and we tried to explain some of the reasons why. Sometimes due to different regression model specifications, differences in just the monitor location can clearly affect the relative risks, different characteristics of the sample population under study and sometimes just random variation. And I'm going to get back to this point in a minute.

When the relative risks of course are small the model specifications can have a very large impact. But

our general consensus was that the body of evidence is too large, the epidemiologic body of evidence, to ignore. And the health endpoints are quite important in terms of the margin of safety considerations.

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OEHHA AIR TOXICOLOGY AND EPIDEMIOLOGY SECTION SUPERVISOR OSTRO: There was a concern about -- some commenters said that we were -- there was a concern about studies relating to ozone exposure when the studies had been primarily focused on PM. And that's basically a result of the PM centric activities that we've been involved in over the last 15 years or so.

So it is true that most of the studies focus on PM. But now there's been more and more over the last couple of years that are ozone specific, as in the recent NMMAPS analysis and now these three other meta-analyses. And many other primary studies are coming out with ozone. We agree with the commenters that it would be nice to have a lot more sensitivity analysis in the primary studies. We can't make that happen, but we can make recommendations and we do engage in conversations with some of the researchers and we will certainly encourage that.

The positive side of that though is if ozone studies are taken out of -- from PM studies, they're less likely to be publication bias. If people are presenting

ozone results because they want to show that the PM results were robust to inclusions of ozone, that means a lot of those studies are included whether or zone is significant or not. So they would be less likely to be publication bias.

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OEHHA AIR TOXICOLOGY AND EPIDEMIOLOGY SECTION SUPERVISOR OSTRO: Regarding the benefit assessment, there was a concern that the Epi studies don't prove causality and that we should rely on the chamber studies for our benefit assessment. And I should acknowledge that EPA did that in 1996 in their risk assessment. They only looked at the likelihood of FEV1 greater than 10 percent and respiratory symptoms that were based on only the chamber studies. Those too require assumptions about the distribution of exercise patterns and time activity and so on. So even using those is not very straightforward.

And I should also acknowledge the Chair published a paper I think in '92, one of the first benefit analyses on ozone where in fact they did that same type of technique. But we should also recognize that those studies drew on papers that were available basically up till -- for the EPA case up till about '94. And Dr. Kleinman's efforts, which I think initially were presented to the South Coast in '89, if I'm not mistaken, so

probably the literature stopping around '88. And as we all know now, there's been several hundred papers published in the last eight years or so that we can now draw on. And I think when you're trying to talk about what the overall population burden is, you definitely want to look beyond the populations that are looked at in chamber studies, that is, healthy young adults.

There's also concern about the ozone mortality that obviously got a lot of attention. And one of the comments that came through often was that since the ozone mortality estimates are variable, they should either not be used at all in our assessment or we should have zero as an estimate from those.

So I think I will move ahead for a second and discuss that -- just to mention another general issue that's been mentioned has been issues relating to our exposure estimation, the rollback method that we used in the benefit assessment, and the averaging time, that is, how we related 1-hour, 8-hour and 24-hour exposures.

So let me just address these in a little bit more detail.

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OEHHA AIR TOXICOLOGY AND EPIDEMIOLOGY SECTION SUPERVISOR OSTRO: So, as I've indicated, the chamber studies are difficult to apply in a population basis. The

Epi studies, in fact, meet many of the criteria that Hill set out in the sixties for causality. But we can also draw on the -- both the human control studies as well as the animal studies to support some causal relationships in some of those relationships.

The methods we used in our benefit assessment are similar to those that the EPA -- U.S. EPA used in their report to Congress. Under Section A12 they do calculations of the benefits of the Clean Air Act, similar to methods used in the regulatory impact analyses that they do for regulations on both -- affecting both particles and ozone, and several published articles that have occurred in the last few years.

And I should note that there was an article just published last week, Environmental Health Perspectives, by Brian Hubble, et al., who he's one of the EPA staff involved in calculating benefits for them. And in his assessment -- or their assessment, they did include mortality estimates as part of their total benefit calculations. Now, they have the disclaimer in the article that doesn't necessarily represent EPA policy. But having worked at U.S. EPA myself for several years, I know it's unlikely that something would have gotten out without going through a rather intensive internal review. So I think it's likely that U.S. EPA will be incorporating

more of these mortality studies in their assessments.

I should also mention that their use of mortality in their benefit assessment was recommended by the Scientific Advisory Board to U.S. EPA, the part of the SAB that reviews the health benefit assessment. And it was recommended that ozone mortality effects be considered for inclusion. And part of the EPA response to that was, "Well, we'll wait and see what these meta-analyses show, these three meta-analyses that we have funded." And as we've heard today, these analyses have now been published -- will be published soon in Epidemiology. And so I would assume that it's likely that subsequent U.S. EPA analysis will in fact include the ozone mortality effects.

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OEHHA AIR TOXICOLOGY AND EPIDEMIOLOGY SECTION SUPERVISOR OSTRO: Regarding the second issue about mortality should be quantified, I think I've dealt with it a little bit. But just to, since it is an important issue, go through it a little bit more, we know now that the Bell analysis, the newest NMMAPS analysis, and several other meta-analyses including those conducted by Jon Levy at Harvard and a WHO analysis that came out a couple months ago, all show positive and statistically significant associations between ozone and mortality. In

some of the studies the associations exist for the full year and for some of the studies for summer only.

A certainty remains of course, and I think will always remain, on what are the proper dose response shapes and what variable should you include in the models and so on. But the uncertainty does imply that it's a zero effect. And, in fact, the heterogeneity and response is not really an unlikely outcome in the epidemiologic literature. And since there was a lot of discussion and presentation of the NMMAPS results, I wanted to just give a couple reasons why there would be variability on a city-by-city basis and why that's not grounds for throwing out the results.

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OEHHA AIR TOXICOLOGY AND EPIDEMIOLOGY SECTION

SUPERVISOR OSTRO: In the recent Bell analysis I think there was actually one city that was negative, all the other cities were positive and a few that were statistically significant.

So some reasons for variability in the response would include actual pure chance, just random events, acts of nature or what have you. You'll just get variability in estimates even under the best of conditions.

But another reason is statistical variation in the procedure. That is, even if you simulated data

showing a relationship between mortality and ozone and you began with a petty good association and simulated the data and then sequentially took out 5 or 10 percent of the data, threw it out and reestimated it, that by itself would give you a large variation in what the estimates would be like. So just having missing days and other issues relating to data and data specification can give you a large variation in results.

Another issue that Dr. Delfino mentioned was the whole issue of measurement error. That is, that for ozone we probably have a lot more measurement error in relating what fix-site monitors are indicating relative to what people are actually exposed to. The issues are much greater there than for, say, particles where we see a lot more infiltrations from fine particles.

So different cities might have different amounts of measurement error based on the spatial pattern of the population and based on housing construction. And we know that in general everything else held constant, that the measurement error would tend to lower the effects observed in the dose response estimates. So certainly that's a good reason for observed variability in the response.

Another reason for some of the NMMAPS results, particularly the lower estimates that were observed, was the actual approach that was used by the NMMAPS review --

the NMMAPS research team. Their initial charge was not to find out the best estimate of mortality that could be used in benefit assessment. Their charge was really to see whether there was an air pollution effect. And so they took a very conservative approach in terms of their modeling.

Specifically, they did things like they had four different variables controlling for weather, they used the same types of model specifications in every city even though we know that there's going to be different effects of weather depending upon location. And, in general, that same approach, that same modeling approach to every city is another reason why you're going to get different variability across the different cities.

Also, we note now that NMMAPS typically gives lower responses -- lower dose response estimates than other efforts. And, again, I think that's related to the conservativeness of their approach.

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OEHHA AIR TOXICOLOGY AND EPIDEMIOLOGY SECTION SUPERVISOR OSTRO: So I'm just mentioning a little bit more about this recent study because it does address some of the concerns of commenters. The newest effort includes 95 cities and another six years of data. Previous efforts included as few as 55 cities and sometimes only several

years of data.

They found associations with both total and cardiopulmonary mortality. They found relatively similar effects for the 55 cities that they used that had full year of data versus cities that only had warm season data and then combining all 95 cities. So the effects were relatively similar.

So this is important. It looks like -- that even though they didn't present effects for the winter only, it looks like if you have effects for the full year and similar effects for the warm season, that it's fairly likely that you might actually see some similar effects for the winter season. I can't vouch for that, but that might be a logical outcome of that.

Also, as was mentioned by some of the commenters, the results were robust to the inclusion of PM10, which was a concern expressed by commenters. And also the issue of confounding of temperature, not only was there a recent paper published in the American Journal of Critical Care Medicine that indicated the temperature was not a significant confounder, but in the Bell analysis they excluded days above 85 degrees Fahrenheit and found very similar effects.

Finally, they found similar effects among the different age groups less than 65 and 65 to 74 and 75 and

above, potentially indicating that it's just not very, very fragile people in the oldest age group that are affected by the inflammatory effects of ozone.

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OEHHA AIR TOXICOLOGY AND EPIDEMIOLOGY SECTION

SUPERVISOR OSTRO: This diagram shows the different meta-analysis that have been conducted over the last couple years. And in the handouts that you have, you have a little bit of detail on each of the different studies. But the first two, the WHO analyses that were published a couple months ago, number 1, is the full analysis and, number 2, is the one that corrects for the potential of publication bias so that they can't really prove that publication bias was there based on their techniques.

Three and four are Thurston and Ito meta-analysis of far fewer studies, but showing very importantly that when non-linear models for temperature were used, the effect estimates for ozone became much larger.

Six and seven are the Bell analyses that we were just talking about. Six is using just a 2-day average of exposure, 0- and 1-day lag for ozone. Seven is using actually a 1-week lag. And actually the effects double when a longer exposure period is used, which indicates that exposures over a longer period of time may be more important for ozone as it is for particles. Most other

studies have not looked at longer term exposure like this. Most have used only 1- or 2-day lag. So that's a rather important finding here.

Then on the far extreme are the Gryparis estimates. And I'm not sure if I'm pronouncing the name of this Greek author. But this is the FEV2 studies that were referred to, study 29 cities in Europe.

And 10 is the full year estimates and 11 is the summer-only estimates.

So here's some details on the models.

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OEHHA AIR TOXICOLOGY AND EPIDEMIOLOGY SECTION

SUPERVISOR OSTRO: But here I've tried to indicate what the percent change -- this is per 24-hour average -- 10 ppb change. And the red dotted line is what we're proposing to use in our benefit estimate as our low estimate. Actually the red lines are off a little bit. But .42 is going to be what we're thinking of for our low estimate, which is half way between what NMMAPS got from their 0- and 1-day lag versus their one-week lag. So we're thinking of maybe probably averaging that estimate. We use about a 1-percent change per 10 ppb 24-hour average, which you see is very close to the center estimate of a lot of the other studies.

And then for the higher estimate, although we're

not catching the real high estimates of some of the other studies, particularly a summer-only study, which might be more relevant for California. But we're still going to be constraining it to be in the range of what some of the other studies have shown, including the Thurston study and the Steeb study. And it's among the higher ranges in the WHO estimate.

So this is our proposed range. And we're attempting here to generate numbers that are reflecting of the uncertainty in the overall estimates.

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OEHHA AIR TOXICOLOGY AND EPIDEMIOLOGY SECTION SUPERVISOR OSTRO: So the third set of comments dealt with methodological issues in our benefit assessment relating to exposure estimation, the rollback methods that we used on the averaging time that we used.

In our exposure estimation we used a process where we attempted to mimic the exposure scenario of the original studies by assigning population to monitors. As I mentioned yesterday, we did that on the county-wide level. And as a sensitivity analysis, we're now going to replicate it and assign exposures to each censor's track in the California cities and see how that changes the results.

We're also -- we also used a proportional

rollback scheme, which was consistent with the way the air quality plans and historical trends have shown ozone to change over time. But we are planning now to do an additional analysis, which would have a less than proportional rollback, to indicate that maybe in some of the areas -- particularly some of the coastal areas the changes in ozone might be as great. So we'll at least look at the sensitivity of that.

Finally, there's a question about our use of national averages of ratios between the 1-hour, 8-hour and 24-hour averages against -- different studies report different averaging times. Since a lot of these studies were conducted all over the world, we use national estimates. But recently we also looked at the California ratios between these. And it turns out the ratios that we looked at for several California cities are very similar to that found for the national estimates and the ones that we used. So it looks like the application of our estimates -- the ratios seem to be pretty good.

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OEHHA AIR TOXICOLOGY AND EPIDEMIOLOGY SECTION SUPERVISOR OSTRO: Before I get to other issues I just wanted to say I think one or two other things about the Epi studies. One, there's a statement made that over -- now the epidemiology is more uncertain than it was 10

years ago. And I have to say I don't think that's necessarily true. With several hundred more studies over several new endpoints, a wide range of endpoints, I think we're certainly less uncertain about the fact that ozone has an impact on public health and not just to healthy exercising individuals.

And the other thing I wanted to just mention briefly was the CASAC process. Again, that was 10 years ago. And I'm guessing -- and I've had some informal discussions with some CASAC members from 10 years ago indicating that they might have different decisions and different opinions if in fact all this epidemiologic evidence was available at that time.

So a couple comments on other health-related issues. One question was that the response of other susceptible populations were not adequately discussed. We did try to include all available information. We've noted now some additional studies that will include in our document but we think we did reasonable job in looking at this susceptible populations in both the chamber studies and in the Epi studies.

There was concern that there was no discussion of the effects of reduced ozone due to historical reductions. And I think the comment here is referring to what people call intervention studies. That is, for particles their

study's now in Dublin and Hong Kong where high sulfur coal was banned. People looked at mortality rates beforehand and afterwards to see whether there was significant changes. And, in fact, did find after the intervention there was very different types of associations.

For the most part, they weren't available for ozone with one exception, which we need to add to the document, which is the Friedman study of the L.A. -- I'm sorry -- the Atlanta Olympics, that Ralph referred to. The changes in ozone on a percentage basis looked like they were the largest. But it was the case that particles and other things did of course change. But there's a very significant change in ozone and in emergency room visits for asthma, where other emergency room visits for other causes were not affected.

So there does seem to be an effect on emergency room visits. We can't say for sure it's ozone, but certainly a leading contender.

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OEHHA AIR TOXICOLOGY AND EPIDEMIOLOGY SECTION SUPERVISOR OSTRO: Also, there was a question about indoor contributions that were not considered, we didn't consider. So our comments, are responses were that ambient air quality standards are for outdoor air. There's a few indoor sources of ozone. And that the Epi

studies basically demonstrate impacts associated with outdoor monitors. One of the nice things about the time series studies is people are followed on a daily basis. And in the panel studies people are followed on a daily basis. And you don't expect a lot of changes to occur on the indoor factors on a daily basis, except the outdoor air that's coming indoors.

So we don't think consideration -- additional consideration of some of the indoor effects would really change any of our conclusions.

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OEHHA AIR TOXICOLOGY AND EPIDEMIOLOGY SECTION SUPERVISOR OSTRO: So that ends my responses to comment.

And Dr. Marty wants to add some comments. But I'm open -- if the panel has other questions that they want me to address, I'm open to answering them at this time.

CHAIRPERSON KLEINMAN: Anybody have specific questions for Dr. Ostro?

Okay. Then Dr. Marty.

OEHHA AIR TOXICOLOGY AND EPIDEMIOLOGY SECTION MANAGER MARTY: Melanie Marty from OEHHA. I just had a couple of comments that came to mind in listening to the discussion yesterday and to the comments today.

One commenter today noted that our -- that the

chamber studies don't really comply with the ATS criteria for adverse health effect. And I would disagree with that. We did in fact see symptoms in the chamber studies, and that was noted in our report.

And also if you look at the description of the ATS criteria for adverse health effects on page B-3 -- it's Appendix B, page 3 -- it includes decreased health-related quality of life issues. And certainly asthma exacerbation falls under that category, which is seen in the epidemiological studies.

And also I would like to note that it also includes a shift in risk factor distribution and, hence, the risk profile of an exposed population, that that should also be considered adverse. And I think that's really important, and possibly response to the concern I've heard Dr. Sherwin express yesterday that you don't just wait until you have a clinically manifest endpoint, that you need to consider the steps leading to that endpoint.

I also heard today that we didn't do a reasonable enough job of describing susceptible subpopulations. And it was I believe described within a couple of context, kids and environmental justice considerations. And we did have some discussion of this also at the workshop -- the public workshop that was held in Sacramento and -- I

forget where the other one was. We do consider at OEHHA that children are a specifically susceptible population with respect to health outcome asthma. And the reasons for that will be described more in this document. I thought they were in there and, in fact, they were not.

Kids have a higher prevalence rate of asthma. Zero to four-year olds have higher hospitalization rates for asthma compared to any other age group including older folks. Kids have smaller airways; so a little bit of airway constriction goes a long way in a kid since resistance is inversely proportional to the cube of the radius.

Other health impacts include school absenteeism, which is partly related to asthmatic exacerbation, but also probably partly related to lower respiratory tract infection. And, in fact, animals studies looking at infectivity models show that asthma increases morbidity and mortality in those -- in those infectivity models. So that's where an animal is exposed to a pathogen. And in this case it was mostly lung pathogens. And you can see whether exposure to ozone makes a difference in the outcome from the animals, and indeed it does.

I did want to also talk a little bit about what we're using, basically chamber studies, to set this standard. But I want to emphasize that we need to

extrapolate that to millions of people in the State of California who will be -- who are exposed and will be exposed. So you're going from a relatively small sample size in the chamber studies to a large extrapolated population.

The chamber studies don't use moderate to severe asthmatics, for ethical reasons. They don't do -- don't use infants. They don't use kids who've had early lung injury. So that we can't just say, okay, .08 is fine. At OEHHA we don't think that that is reasonable. And that is one of the reasons we've looked at 70 parts per billion as the top end that we're willing to go with.

And, finally, I think it hasn't been emphasized enough, at least -- it has in our report but not necessarily in the discussion -- that the toxicology -- the animal toxicology data strongly support the adverse effects seen in humans. You see airway inflammation by a number measures. You see epithelial injury. There's adverse effects on lung development when you're looking at primate models. And there's also evidence for enhanced response to allergens and conditions that predispose to asthma in animal models.

So I think these are important things to think about, that ozone-induced toxicology is qualitatively similar across species, rodents and primates. And also --

if you look at the human data, it's also totally in concordance with what happens in people.

Thank you.

CHAIRPERSON KLEINMAN: Thank you.

It's 12 o'clock. We really have received a lot of information kind of on the fly this morning. And I wanted to propose the following: That we take a 15-minute break so people can freshen up a little bit and check out if necessary; that we reconvene at about 12:20.

And during that period I'd like to have a very brief executive session with the Committee. We had a very intense discussion last night after the meeting, going over remarks and comments and all of the written material. And so the question would be: Has anything that occurred today raised questions that we need to discuss more thoroughly? Are there other questions that we need to ask of the staff while we have them here to clarify any remaining issues?

And if not, what I would propose to do is present some preliminary recommendations from the Committee and will adjourn the meeting probably by one o'clock rather than stopping and having lunch.

Now, if there's a -- you know, if anybody's got a real serious objection to that, you know, we could --

ADVISORY COMMITTEE MEMBER SHERWIN: I'd like to

add one thing before we quit, if we can. The subject of ATS came up several times. Delfino talked -- Dr. Delfino talked about it. Dr. Marty talked about it.

Dr. Gelfand relied upon symptoms. And that of course is what comes out of the ATS. And I want to make it clear that I seriously objected to the ATS when it first came out. I was invited to be a part of it. And I declined because I couldn't convince people that morbility was the area -- you had to get away from symptoms. You can't wait -- it's like cigarette smoking, you can't wait for the cancer to develop before you start talking about the adverse effects.

So what I am proposing, I'd like to give more emphasis to this. And the fact is that I actually published an article in Environmental Health Perspectives in 1983, two years before the Thoracic Society's statement. And I'd like to enter that into the record, say that this is -- a lot of things have happened since then that substantiate it more. I mean there's a -- enhance it or make it even more pertinent. But even then, in 1983, I think it's highly relevant. And so it is in Environmental Health Perspective, Volume 52 -- let's see, I had it down as pages 172 to 182, a 1983. And I'll leave a copy with you if you like.

ADVISORY COMMITTEE MEMBER DELFINO: I think they

addressed that by saying that the importance of increased risk factors were in some cases as important as things like symptoms and that risk factors can predict serious morbidity. And we could add to that, for instance, inflammatory markers like C-reactive protein or as predictive of myocardial infarction as cholesterol.

ADVISORY COMMITTEE MEMBER SHERWIN: But they discussed those biomarkers. And they said, "Well, until we show some definitive tie to them" -- that was in the update -- "we really can't do much with them." And the bottom line says when you people used it, when Dr. Gelfand used it, when almost everybody else used it, if it doesn't do something, you can see clinically it isn't significant. And that's the message I think I'm trying -- I was trying to get across, that that's -- we've got to start talking about subclinical effects of the emphysematous person who loses 70 percent of his or her lung before anybody even knows it.

And pulmonary function tests -- I've asked this to chest physicians and no one's ever challenged me on it. I said you -- from my studies with autopsies and your clinical correlations, I believe you have to lose about 25 percent of your lung before your functions has first become positive. Now, that is a one shot, not follow-up.

So you're either pulmonary function test on

somebody or you're not. And if they'd lost 25 percent of his or her lung, it's going to be iffy to detect.

So that's the message.

CHAIRPERSON KLEINMAN: Thank you.

One other thing. I think -- Sue, were you able to get some cookies for sustenance?

We have them. So -- we're not heartless. So what I'd propose is we break until -- well, it's 10 after now. So let's give it till 12:30 and reconvene at 12:40 for a brief presentation of our findings.

ADVISORY COMMITTEE MEMBER HAMMOND: Is that executive session?

CHAIRPERSON KLEINMAN: We'll try to meet before that.

(Thereupon a recess was taken.)

CHAIRPERSON KLEINMAN: Okay. If everybody can find their seats, we will reconvene.

(Thereupon an overhead presentation was Presented as follows.)

CHAIRPERSON KLEINMAN: I want to put -- just to put this back in context in case people here weren't here yesterday, the background of this process is to some extent based on the Children's Environmental Health Protection Act that required a review of air quality standards to make sure they were protective of susceptible

populations including infants and children, that there was an adequate margin of safety. And as part of that process, the review of specific pollutants were prioritized for a full reanalysis. And this is the second of those reviews, the first being PM.

So the review of ozone was a promulgated -- or not promulgated -- but instituted in response to this, as well as it being overdue actually for review anyway. We are supposed to review these things every five years, and we have been somewhat remiss.

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CHAIRPERSON KLEINMAN: Five points were examined by the individuals who looked at the issue of whether ozone per se -- the ozone standard was protective of children. And they looked for evidence of effects at or near existing ambient air quality standard, which at the time was a 1-hour standard at .09 ppm. They looked at the nature and severity of effects, magnitudes of risk. They looked for evidence that children may be more susceptible than adults. And they looked for the degree of outdoor exposures relative to the level of the standard. And as part of that SB 25 review they identified clinical and epidemiological studies, which did demonstrate effects of ozone on pulmonary function, asthma exacerbation, mortality in children -- morbidity rather in children and

adults at or below the 1-hour California standard.

The review concluded with the feeling that there was a need for more stringent standard, which -- or a different averaging time or both. And the current staff recommendations have taken that to heart.

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CHAIRPERSON KLEINMAN: The staff has done a tremendous job. They've done a very comprehensive review of relevant studies. And the interpretation and analysis of the very large base of data has been -- is remarkable. But the Committee does have some suggestions for additional studies that should be included in the staff report. And those will be provided on a chapter-by-chapter basis.

And the additional information that we're recommending are not in the -- are not going to negatively impact the current direction of the staff recommendations. They are supportive and they do provide additional information and basis for developing the standard recommendations.

We do want to say as a committee, in looking at the various studies that were cited in the staff report, when you look at any individual study, that you can find limitations and reasons why there are inadequacies; but when you look at the aggregate of the studies in sort of

an integrative way, they do support the relevance of the standards, and the evidence does appear to have coherence.

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CHAIRPERSON KLEINMAN: The staff looked at susceptible populations. And they've appropriately identified children, outdoor workers and athletes who are out of doors often during periods of photochemical activity in the summer months, and the rest of the year as well, and who are often doing exercise or other activities that would raise their ventilation rates and increase their exposures in dose. Also individuals with airway allergies appear to be susceptible populations.

Other populations that should be considered: COPD and cardiovascular patients have been studied. There are very few real studies of these populations. Most of these studies have relatively small numbers of subjects involved. The data are suggestive and do follow the pattern also showing adverse effects of ozone. But the sample size has not been large enough to achieve statistical significance. This is an area that probably should receive additional attention in the future.

Are data on infants and children appropriately considered? The Committee's feeling is that there are several areas that have been not studied extensively, including in utero exposures and exposures of neonates.

And there are databases that are now beginning to get published that will need to be considered in future reviews of the ozone standard.

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CHAIRPERSON KLEINMAN: The studies all have uncertainties. For the health effects studies, the staff has incorporated discussions in the specific descriptions of the studies. There are some limitations of the various types of studies. And it might make it easier to put those in context if they were collected into a section that dealt specifically with study limitations and the impact of those limitations on the conclusions.

In terms of monitoring and background, we've heard a great deal of that today. I think -- and yesterday as well. There are issues of measurement precision and the relationship of the measurements to the not-to-be-exceeded designation that should be more clearly explained in the section on monitoring. The chapter is rather terse and there are -- it is an important issue, not so much in terms of setting the standard, but in making sure that the people who developed the implementation rules will be able to interpret the recommendation properly and make sure that if they do propose regulations, they can meet a not-to-be-exceeded designation.

The method for differentiating exceptional events that we briefly heard mentioned this morning is still not easily understood from the chapter. And I think a little bit better explanation of that process would be very useful.

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CHAIRPERSON KLEINMAN: The Differences in patterns of exposure for various types of susceptible populations are briefly discussed, but could be expanded especially in terms of children and infants. And there have been some studies done that provide some time activity information, and that could be presented as part of the staff report.

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CHAIRPERSON KLEINMAN: So the staff recommendations were to retain ozone as an indicator for oxidant pollutants. And the Committee feels that the monitoring method does not truly measure some of the other oxidant gases; and the retention of ozone as an indicator for oxidant pollutants would only be appropriate if we know for sure that ozone's a good surrogate for these other oxidants.

However, ozone as a designated pollutant is certainly appropriate and, especially given the degree to which the chamber studies in which ozone is clearly

measured by the same techniques we're using in ambient monitoring, makes it very clear that what we are talking about here in terms of the health-based standard is really the health effects of ozone.

And that does not eliminate the possibility that it may need to be looked at in the future that there are other oxidants and very little is known about their specific health effects and whether or not they contribute to the perceived effects of ambient ozone.

So the staff has recommended retention of the 1-hour ozone standard at .09 ppm, they propose a new 8-hour average ozone at .070 ppm, and that the designation is not to be exceeded.

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CHAIRPERSON KLEINMAN: The Committee has some concerns that although the proposed standards which meet the review of the AB 25 panel, which indicated that we needed to have additional stringency in the regulations, where we are concerned that there may still be effects in susceptible populations since the chamber studies at 6.6 hours demonstrated effects at .08 ppm -- and those were 6.6 hour exposures -- an 8-hour standard at an average of .07 ppm gives you a higher integrated exposure. And so -- and we don't have any studies at .07 ppm. However, there are some data from individuals who did respond to .06 ppm.

And presumably Dr. Adams will get that paper published soon.

(Laughter.)

CHAIRPERSON KLEINMAN: But given the importance of the 6-hour studies and the setting of the standard, the Committee would like additional justification for the differentiation between an 8-hour standard versus a 6-hour standard.

The benefits chapter suggest significant health as well as monetary effects -- or monetary benefits. But the focus was on the monetary benefits. Some of the limitations of the epidemiological studies were presented as part of the benefits analysis, and those limitations should be brought up into the chapter on epidemiology in the main part of the report as well.

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CHAIRPERSON KLEINMAN: Future research I think is essential. There are areas in monitoring, especially in the area of determining what personal exposures are to ozone. We really don't have good techniques for doing this. But we've got great engineering talent in California that could develop methodology, and that should be encouraged.

We should look carefully at the other oxidant gases, things like peroxides and other oxidants, that

might have health effects, that would be present and might be present at the same time as ozone and might contribute to the ambient effects.

In terms of health studies, we definitely should encourage studies of other susceptible population groups, including COPD and cardiovascular diseased individuals.

We need to look at new indicators of biological response both in mechanistic terms as well as in being able to use these new methods and new responses in determining whether there are adverse health effects.

And although we use pulmonary function to a great extent, the links between changes in pulmonary function and development of long-term disease is an area that does need, you know, encouragement for research. It's an area that intuitively it seems to make sense, that there should be a connection. In the animal studies, with non-human primates we see a connection between loss of bronchiolar tissue and changes in lung architecture that would seem to also relate to changes in pulmonary function. And so pulmonary function I think is a very important indicator and should be evaluated more closely in terms of how it relates to frank disease.

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CHAIRPERSON KLEINMAN: So the preliminary recommendations of the Committee are that the staff

recommendations to retain the 1-hour standard and institute the 8-hour standard .07 ppm be accepted; that the document -- the staff report recognize that the ozone monitoring may not measure other oxidant gases; and that the total oxidant content of the atmosphere may be higher than measured by the UV method.

Ozone studies in the future should receive research support to expand or replicate key findings that could modify how we look at the adequacy of the margin of safety. And specifically studies involving in utero exposures, neonates and better monitoring techniques should be part of the package.

We also strongly recommend that over the next five years research be encouraged to answer some of the key questions, and that we do not allow the standard to be left as is, that it be re-reviewed in five years. This was really a recommendation made in the staff report. And we want to very strongly endorse that concept, that ozone is an important pollutant, that we do need to look at it in the light of new data. And as we've seen, the amount of new information has been phenomenal over the last six years -- or actually nine years, I guess, almost. And over the next five years I expect that there will be a lot more significant results that may make us want to reevaluate the standards on a continuing basis.

So that's where I'll stop. If there are any questions for us from the staff, any specific things that we've not made clear yet --

HEALTH AND EXPOSURE ASSESSMENT BRANCH CHIEF BODE: Mark, do you have any questions?

No, we have no questions.

CHAIRPERSON KLEINMAN: Okay. So --

HEALTH AND EXPOSURE ASSESSMENT BRANCH CHIEF BODE: Do you want me to kind of talk about the process we're going to do?

That basically we really anticipate getting your final findings. Once we get the Committee's final findings, we will then modify the staff report to respond to your comments and your recommendations. And then at that time we'll make the report available for a 45-day comment period prior to a hearing before the Air Resources Board, in which we anticipate hopefully for an April Board meeting. But we'll see how large the task is to modify this document.

And then also we plan to hold some public workshops as well prior to that board meeting to talk about changes we've made to the document.

So with that, I'd also like to thank, Dr. Kleinman, you and all your Committee members for all the time you've taken -- not just reviewing the document -- in

the last two days from your business schedules, and we very much appreciate all the time.

CHAIRPERSON KLEINMAN: Well, I'd like to thank the Committee members for putting up with this arduous schedule that we've kind of worked through. I'd like to thank the public for their very considered responses and comments. And they are being evaluated as part of the package. And I'd like to thank the ARB and OEHHA staff for doing a tremendous job of summarizing a very large and interesting diverse set of literature.

So with that, I will close this session.

We're adjourned.

(Thereupon the Air Resources Board, Air Quality Advisory Committee adjourned at 1:05 p.m.)

CERTIFICATE OF REPORTER

I, JAMES F. PETERS, a Certified Shorthand Reporter of the State of California, and Registered Professional Reporter, do hereby certify:

That I am a disinterested person herein; that the foregoing California Air Resources Board, Air Quality Advisory Committee meeting was reported in shorthand by me, James F. Peters, a Certified Shorthand Reporter of the State of California, and thereafter transcribed into typewriting.

I further certify that I am not of counsel or attorney for any of the parties to said meeting nor in any way interested in the outcome of said meeting.

IN WITNESS WHEREOF, I have hereunto set my hand this 26th day of January, 2005.

JAMES F. PETERS, CSR, RPR
Certified Shorthand Reporter
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